DISEASES *of the* CHEST

VOLUME IX SEPTEMBER-OCTOBER, 1943 NUMBER 5

Bronchogenic Carcinoma*

FOSTER MURRAY, M.D., F.C.C.P.
Brooklyn, New York

INCIDENCE

Increasing General Incidence—It may be safely stated that there has been a definite and appreciable increase in the occurrence of primary malignant tumors of the lung, the vast majority of which are now recognized as true carcinomas of bronchogenic origin. Despite past controversies, there is fairly universal agreement among pathologists and clinicians who have explored deeply into the subject that this increase is actual and not merely apparent or relative. Improved diagnostic methods may adequately explain our more frequent recognition of the presence of these neoplasms ante mortem, but it is also a fact that post mortem histological findings have yielded a considerably higher percentage of such cases in the past 15 to 20 years. Perrone and Levinson[1] claim that 10 per cent of all carcinomas at autopsy are found in the lungs, second only to carcinomas of the gastro-intestinal tract. Other statistical studies do not support quite so high a rating. Menne and Anderson[2] review very thoroughly this most interesting subject and furnish a complete bibliography. It may well be that the lengthening span of life plays a role in this general increase.

Age Incidence—These tumors occur predominantly between the ages of 40 and 60 years. Of course, they do occur in earlier and in later years (three in our own series of 20 cases were under 40 years of age), but these are exceptional. This age bracket (40 to 60 years) is well to remember, for the symptoms of this disease often closely simulate those of pulmonary tuberculosis, of which the predominant age incidence is between 15 and 45 years. That fact alone should put us on the alert to suspect malignancy in the older age group, where consideration of symptoms alone might lead us astray.

Sex Incidence—A composite of all statistics taken from the litera-

*Presented at the Meeting of the American College of Chest Physicians, Atlantic City, New Jersey, June 8, 1942.

Copyright 1943 by the American College of Chest Physicians

ture places the sex incidence predominantly with the male, in the ratio of about 8 to 1.

Frequency of Occurrence by Lobes—In the order of decreasing frequency, pulmonary malignancies involve (1) right upper lobe, (2) left upper lobe, (3) right lower lobe, (4) left lower lobe.

PATHOLOGY

Origin—It is practically unanimously agreed by pathologists, in the light of present-day knowledge, that carcinoma of the lung originates from the basal cell layer of the bronchial mucosa, regardless of whether the tumor is centrally or peripherally located. It may spring from a major stem bronchus or from one of its many branches, including the finer terminal ones. Ewing[3] described a type of carcinoma arising from the alveolar epithelium, but this is admittedly of very rare occurrence. The so-called diffuse carcinomatosis of the lung, resembling miliary tuberculosis on roent-

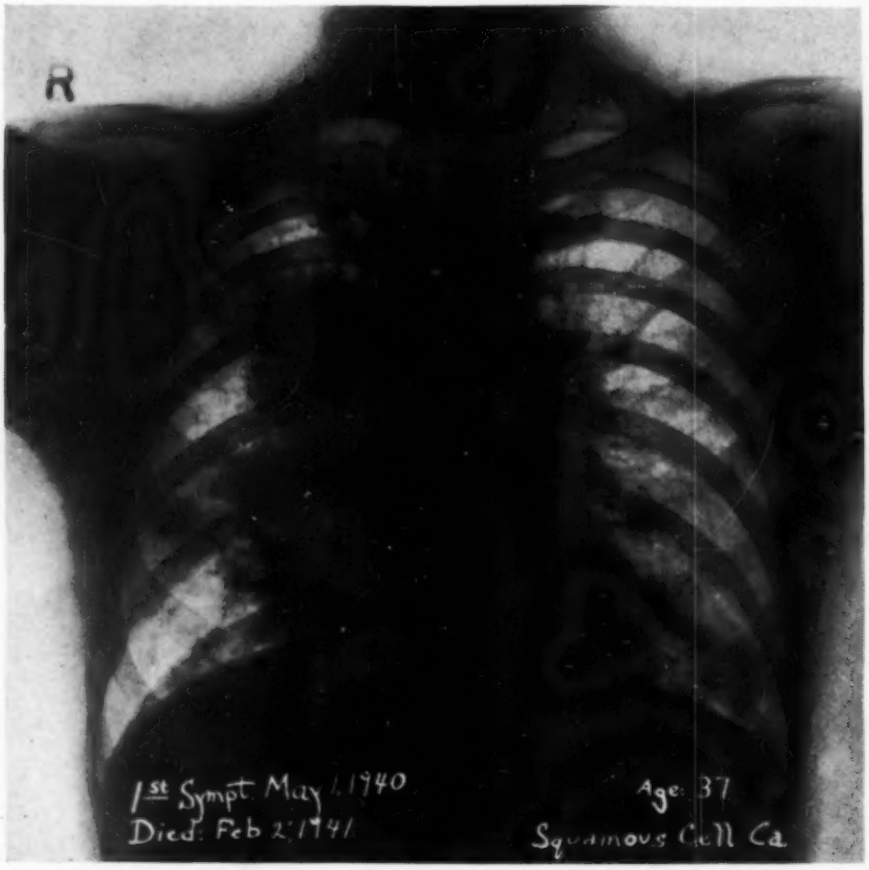

Fig. 1—Bronchogenic Carcinoma of squamous cell type. Death resulted from suppurative pneumonitis. Note mass projecting out from the right hilum with evidence of metastases in same and in contralateral lung.

genography, is in all probability not primary cancer of the lung, but evidence of metastatic deposits of some remote primary tumor.

The superior pulmonary sulcus tumor, first described by Pancoast in 1924, is now generally recognized as a carcinoma of bronchogenic origin though of somewhat atypical behavior. It is to be found in the extreme vault of the thorax and tends for this reason early to erode the bony structures which invest it. Pain is usually the presenting symptom—pain about the shoulder, down the arm, extending sometimes even to the fingers. This is followed by atrophy of the muscles of the forearm and the hand. Due to pressure on the cervical sympathetic a typical Horner's syndrome is apt to appear. Death usually occurs within a year of the presenting symptom.

TYPES OF BRONCHOGENIC CARCINOMA

All types of bronchogenic carcinoma display the characteristics of malignant tumors found elsewhere in the body. They are invasive,

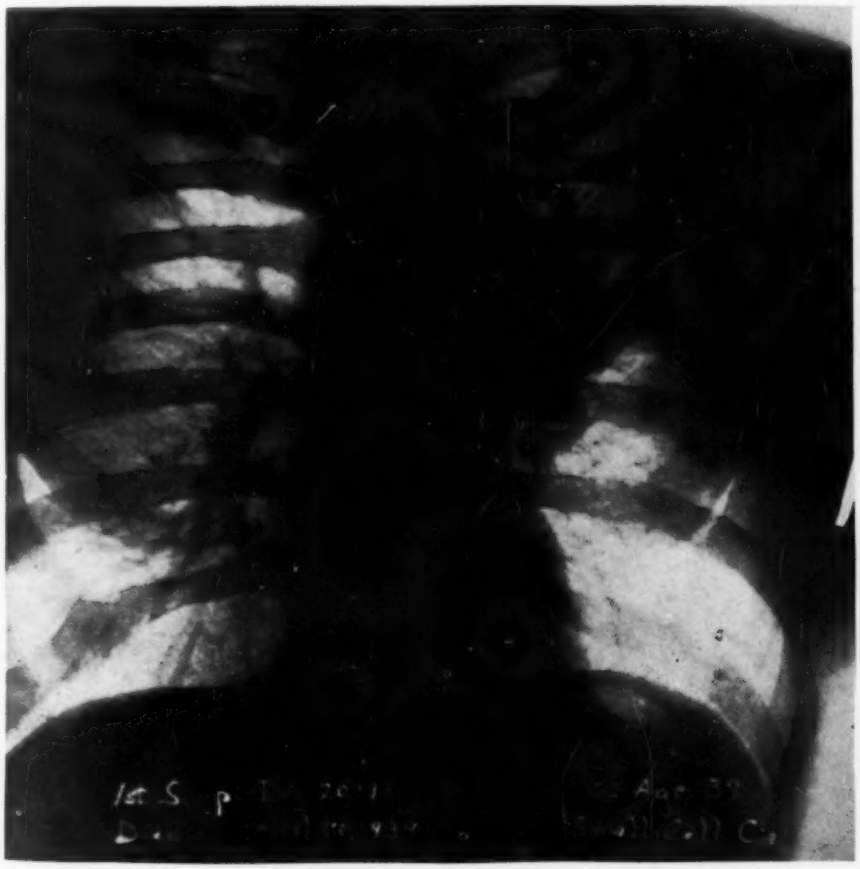

Fig. 2—Bronchogenic carcinoma of small cell type. Note broad base of mass projecting out from the left hilum with strands of infiltrating tissue radiating out into the lung parenchyma.

destructive, obstructive; they tend to ulcerate and to erode, to become secondarily infected and to suppurate; and they metastasize. They vary in their degree of malignancy and so may be graded according to the scale worked out by Broders, the more anaplastic among them being characterized histologically by an increase in chromatin content of nucleus, marked evidence of mitosis, loss of cellular polarity, and more intensive and accelerated invasive properties. Their behavior is conditioned by their basic cell type, which may be conveniently divided into three groups—squamous cell, small cell, and adenocarcinoma, in order of frequency of their occurrence. All follow the same pattern of malignancy but differ one from another in accentuation of certain individual features.

The squamous cell type, also known as epidermoid and epithelioma, comprises about 42 per cent of all bronchogenic carcinomas. It may occur anywhere along the course of the bronchial tree but usually springs from a point within or near the *large*

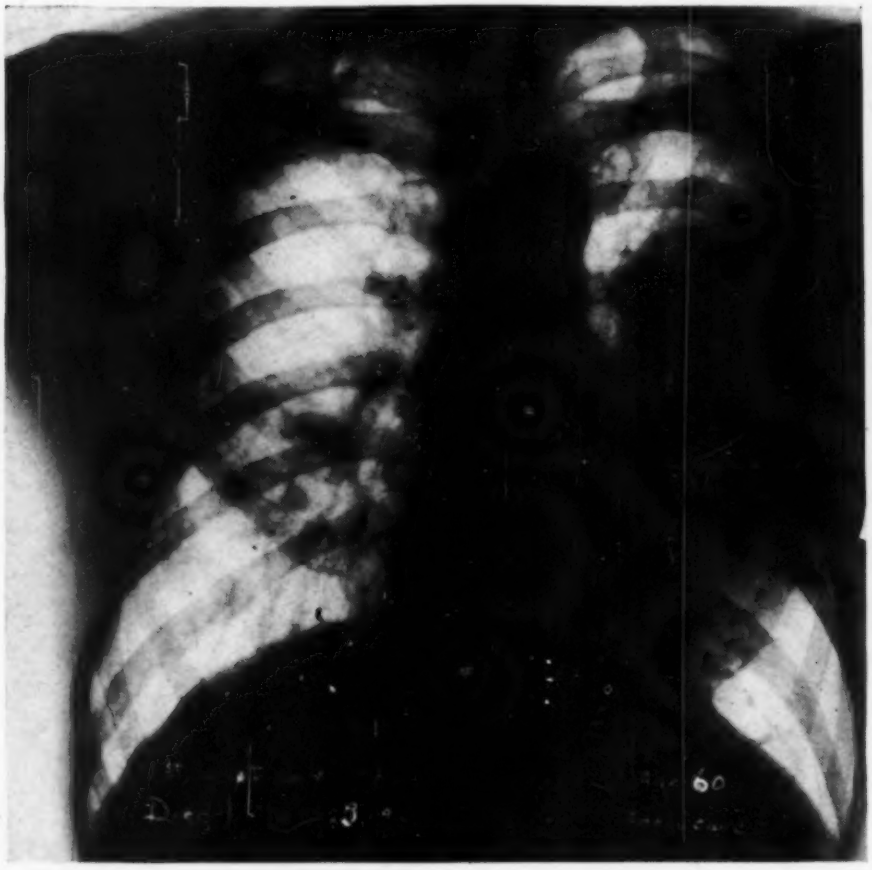

Fig. 3—Bronchogenic carcinoma of adenocarcinoma type. Note area of normal aerated lung separating mass from the left hilum and evidence of metastases in contralateral lung.

branches of the stem bronchus. It evolves more slowly than the other types and tends to metastasize a little later; hence is *relatively* less malignant. Developing more slowly, it brings about a more gradual atresia of the portion of the bronchus it attacks, creating in its evolution all the so-called check-valve phenomena so clearly described by Chevalier Jackson.[4] Secondary infection and suppuration and atelectasis of that portion of the lung distal to the obstruction occasioned by the growth occur more frequently in this type. Death results more often from these complications than from the essential malignant properties of the growth itself.

The small cell type, including the undifferentiated cell type, the oat cell, and the transitional cell, comprises about 33 per cent of all bronchogenic carcinomas. This is by all odds the most malignant, as it is the most anaplastic of all pulmonary neoplasms. Tumors

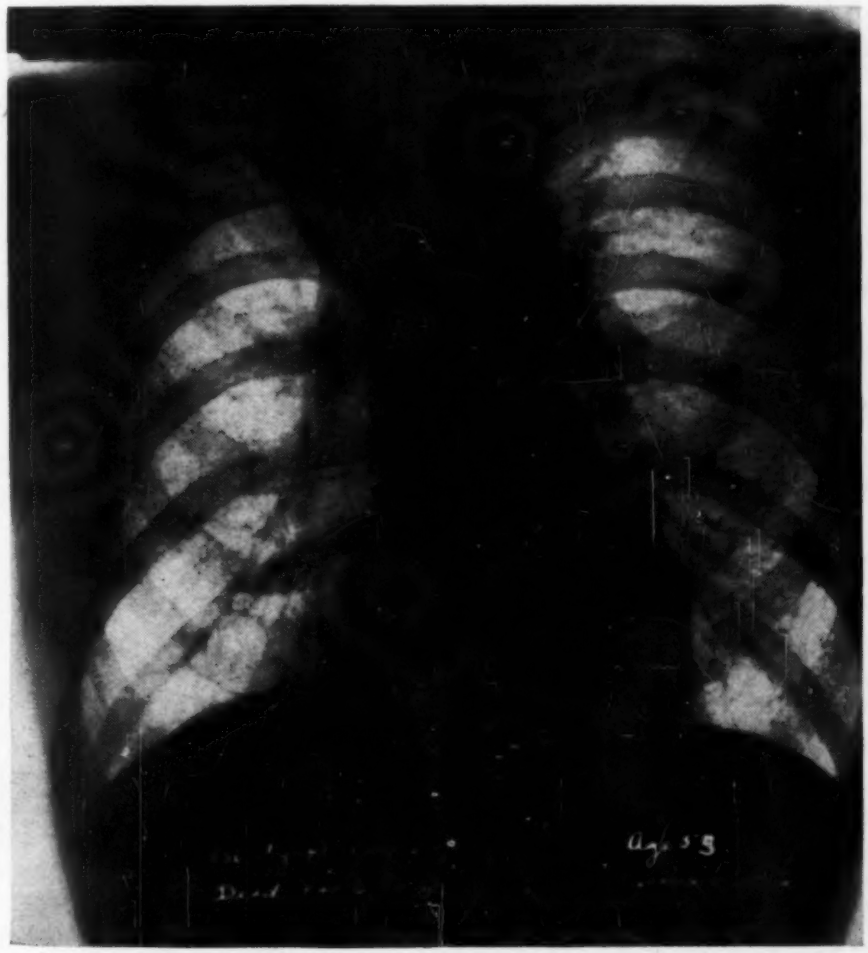

Fig. 4—Bronchogenic carcinoma of squamous cell type. Note atelectasis of right upper lobe with clear cut line of interlobar fissure.

that were previously considered to be sarcomas are now included in this group. While it, too, may arise from any part of the bronchial tree, it usually is found either close to or within the *major stem bronchus*. It is vigorously invasive, metastasizing most frequently by way of the lymph stream directly to the tracheobronchial and paratracheal lymph nodes. It grows with great rapadity and may early give rise to evidences of pressure on the superior vena cava, the recurrent laryngeal nerve, the phrenic nerve, or the esophagus. It causes death, usually before secondary suppuration or pneumonitis intervenes.

The adenocarcinoma type comprises about 25 per cent of the malignant tumors of the lung. It is more often *peripherally located* though it may arise from one of the larger branches of the major bronchi—practically never from the stem bronchus itself. In its earlier stages of development there is an appreciable gap between it and the hilum, though the gap may lessen considerably as the mass enlarges. It metastasizes somewhat earlier than the squamous cell type and chiefly, though not wholly, by way of the blood stream, situated as it usually is in the more highly vascular part of the lung. Metastases to the brain and other remote structures occur more frequently (in this type) and give rise to symptoms often before the

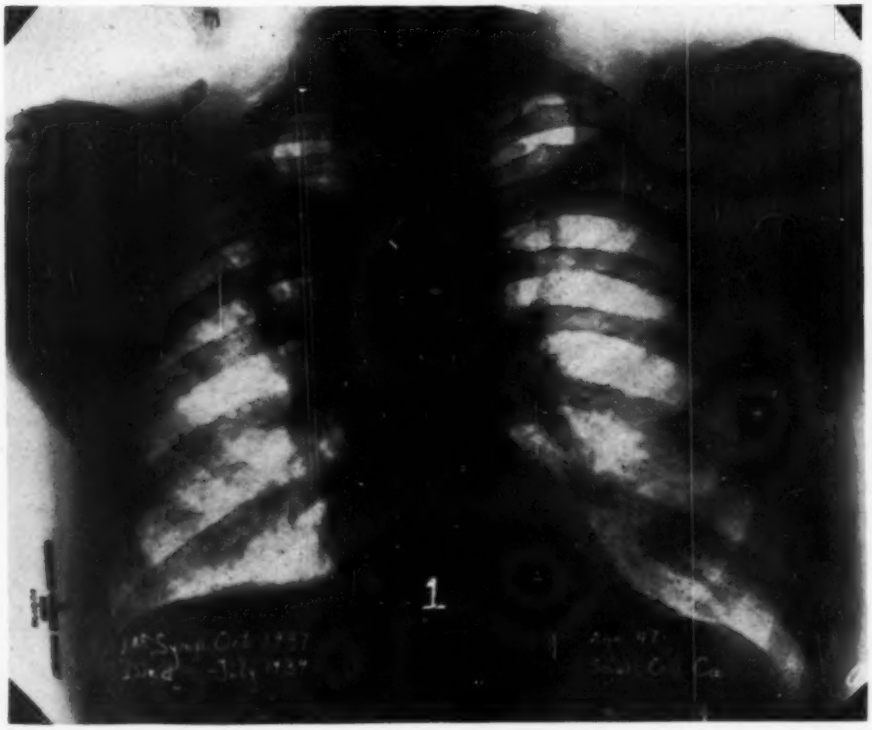

Fig. 5A—Small cell bronchogenic carcinoma of right major ascending bronchus with excavation within it.

parent growth itself does. Pleural irritation and consequent development of effusion occurs more often in this type than in the others. In degree of malignancy it occupies a place between the squamous cell and small (transitional) cell types.

Gebauer[5] affirms that these three types may be fairly accurately differentiated by clinical, radiologic, and bronchoscopic study even before histological examination is undertaken. We have learned much in our study to support his stand. One should not, however, attempt to make a final or positive diagnosis on this basis. In observing a series of pulmonary neoplasms the occasional nonconformist type will be encountered, whose clinical course and biologic evolution will be unpredictable and whose histological findings *at autopsy* may even reveal an admixture of several types in the same mass.

Metastases—All types of bronchogenic carcinoma metastasize

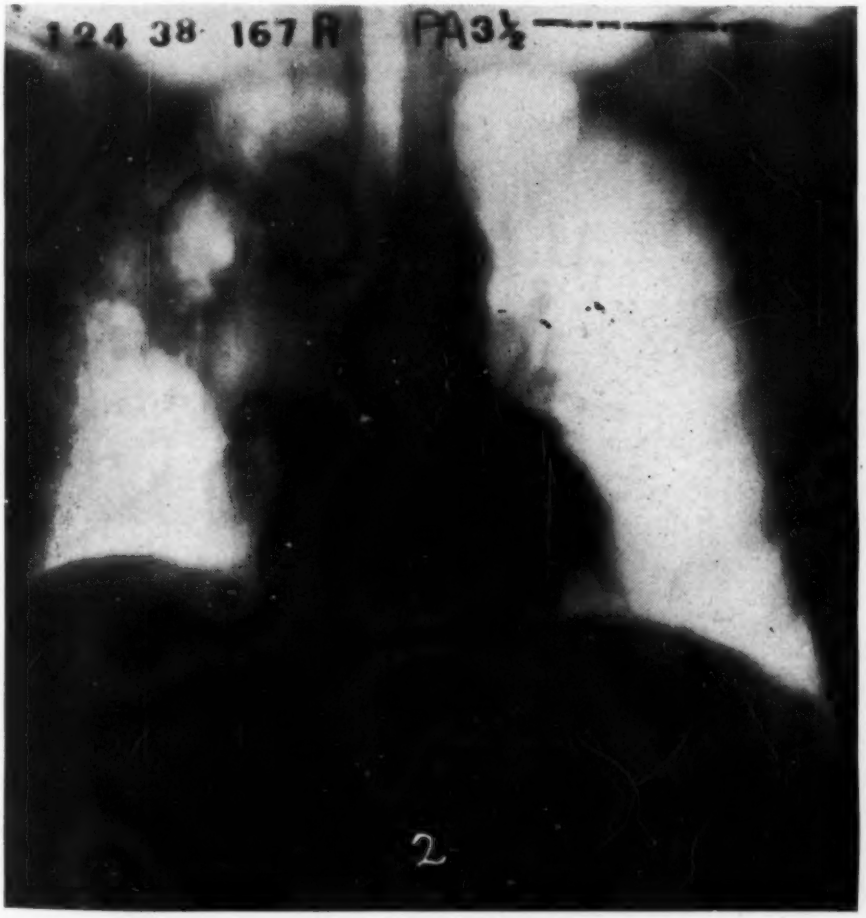

Fig. 5B—Tomographic film of small cell bronchogenic carcinoma of right major ascending bronchus with excavation within it.

freely by way of both the lymph stream and the blood stream. The small cell type does so early and vigorously, predominantly by the way of the lymph stream, less often by the blood stream. The adenocarcinoma does so early, too, preponderantly by the blood stream, to a lesser degree by the lymph channels. The squamous cell type is definitely slower in metastasizing and does so fairly equally by both routes.

The site of metastases in the order of frequency are as follows:

1) Regional Lymph Nodes—Tracheobronchial, paratracheal, supraclavicular, cervical, axillary.

2) Liver, lungs, bones, kidneys, adrenals, pleura, brain, female pelvic adnexa.

ETIOLOGY

Aside from the knowledge that some form of chronic irritation plays a leading role in the etiology of these tumors nothing really definite is known. Much speculation has been indulged in but nothing actually proven. Simons[6] reviewed exhaustively all possible etiological factors, pointing out that some form of chronic irritation

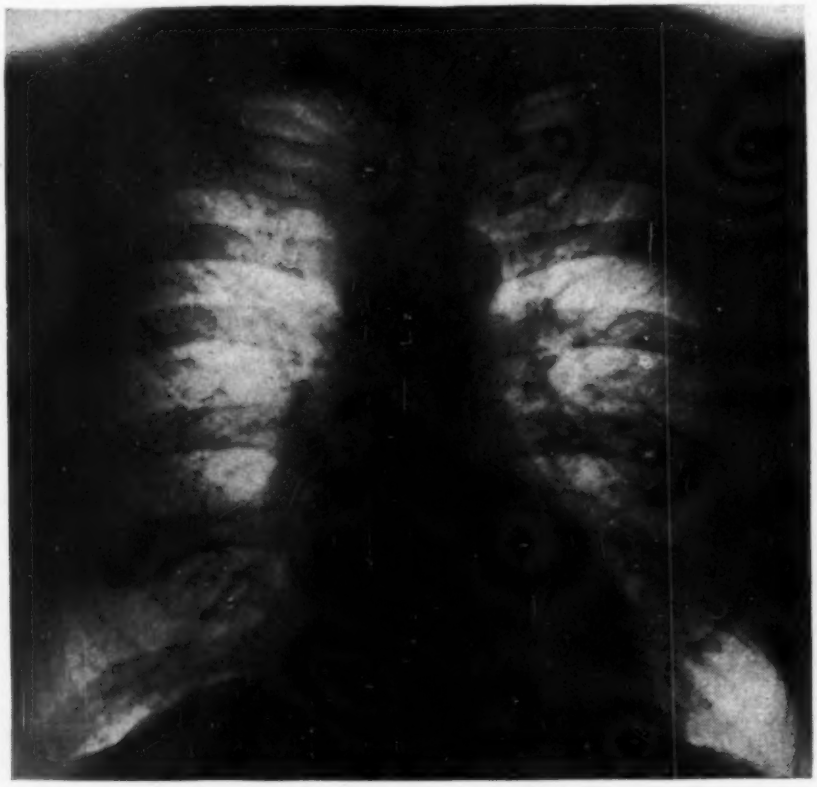

Fig. 6A—Stage in the evolution of an adenocarcinoma of the lung of rather long duration. Small mass at right base, May 18, 1928.

underlay practically all of them. With slight rearrangement these etiological factors are as follows:

1) *Chemical*—Inhalation of (a) tar particles, (b) motor exhaust fumes, (c) war gases, (d) tobacco smoke, (e) certain dusts (as in pneumoconiosis or in the case of the Schneeberg miners). It is assumed that there has been a marked increase in the above during the past 30 years, coincident with the increase in the incidence of bronchogenic carcinoma.

2) *Mechanical*—Trauma (19 out of 500 cases).

3) *Bacterial*—(a) Tuberculosis 15 per cent, (b) influenza (again the increased incidence since the great influenza pandemic of 1918-1919 has been noted), (c) chronic respiratory infections and their sequelae—lung abscess, chronic bronchitis, bronchiectasis (many believe these latter the result rather than the cause).

4) *Heredity*—64 out of 930 cases.

All the above may be vital contributing factors but none of them by themselves have been proven conclusively to have produced cancer of the lung. Apparently some constitutional defect, probably

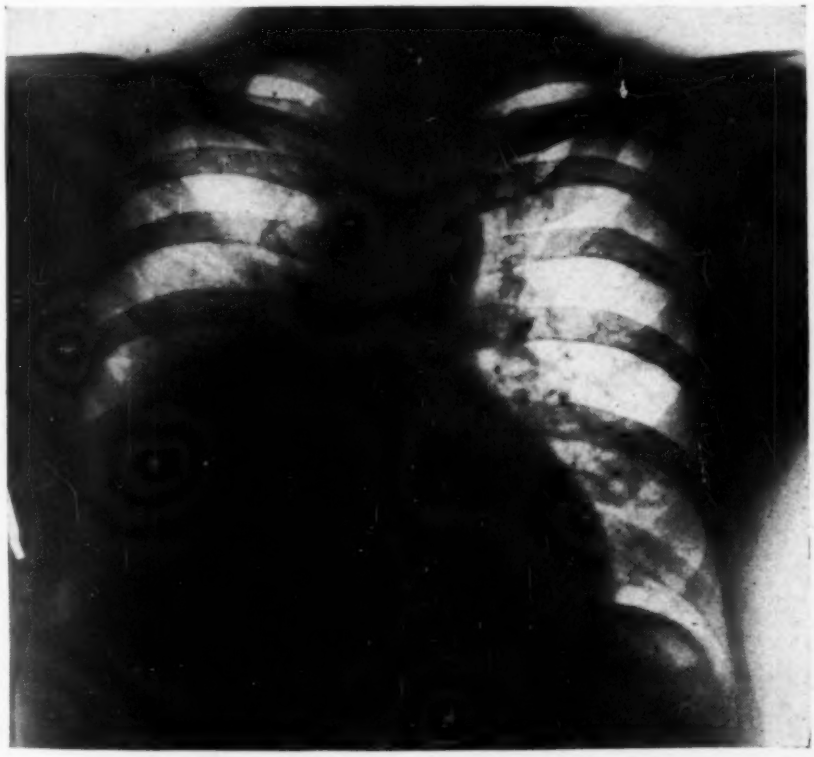

Fig. 6B—Stage in the evolution of an adenocarcinoma of the lung of rather long duration. Same case on November 20, 1929, showing the mass plus collateral pneumonitis.

congenital, must be present to pave the way for the deleterious effects of chronic irritation.

SYMPTOMS

The disease in its onset is characterized by symptoms so protean and dissembling in nature as rightly to merit for it the term applied by Overholt, "The Great Masquerader of Pulmonary Diseases." Usually the onset is insidious, conditioned largely by the location and size of the growth as well as its basic type. In about one-third of the cases symptoms arise more suddenly, when the disease may be mistaken for "acute bronchitis" or "pneumonia."

Arranged as nearly as possible in order of frequency, the predominating symptoms are:

1) *Cough and Expectoration*—The cough at the beginning is apt to be dry and spasmodic, the growth at this time acting as a foreign body in the bronchus. The irritation reflexly stimulates the mechanism of attempted expulsion; hence the spastic quality of the

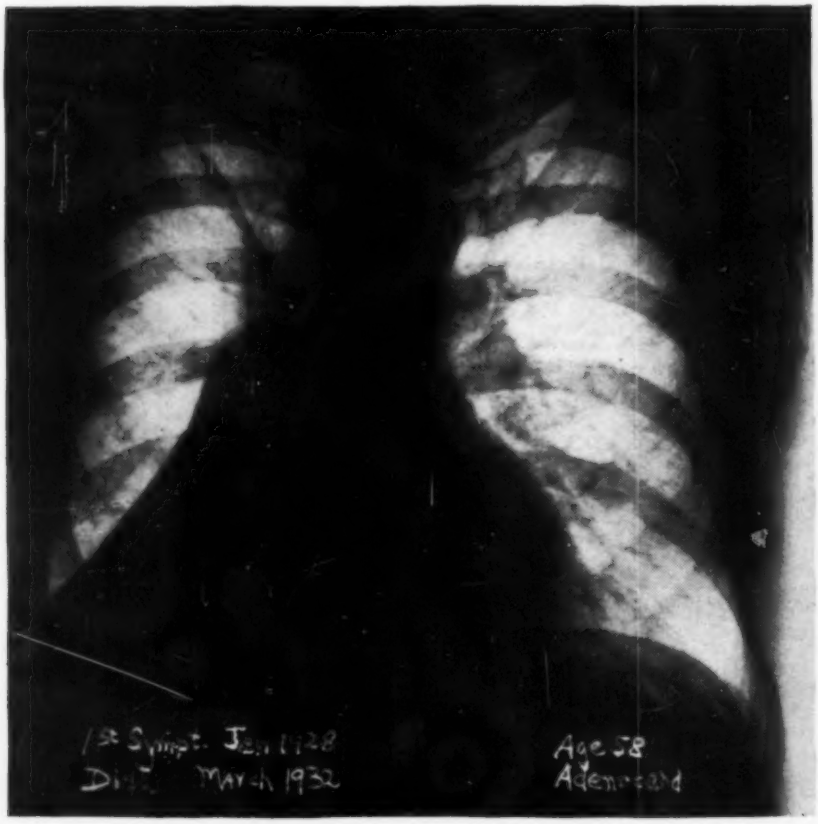

Fig. 6C—Stage in the evolution of an adenocarcinoma of the lung of rather long duration. Same case on May 9, 1931, showing atelectasis of right lower lobe. Patient, age 58, died in March, 1932, following a massive haemoptysis.

cough with little or no sputum. Later, however, gradual atresia of the bronchus interferes with drainage distal to the growth, with backing up of infection and eventual development of suppuration, pneumonitis, and atelectasis. Cough then is followed by expectoration which is scanty at first, later more profuse, and often, though not always, purulent. Cough and expectoration occur in about 65 to 70 per cent of all cases.

2) *Pain*—Few if any cases occur without this symptom arising at some time during the progress of the disease. Most often it is early and constant. It frequently interferes with sleep at night. It is boring in quality and invariably found on the same side as the growth. It is not affected by deep breathing unless occasioned by complicating pleurisy, in which event the pain is likely to be sharp and stabbing in character.

3) *Dyspnoea*, occurring in about 60 per cent of all cases. It is occasioned by varying degrees of intrabronchial obstruction. When

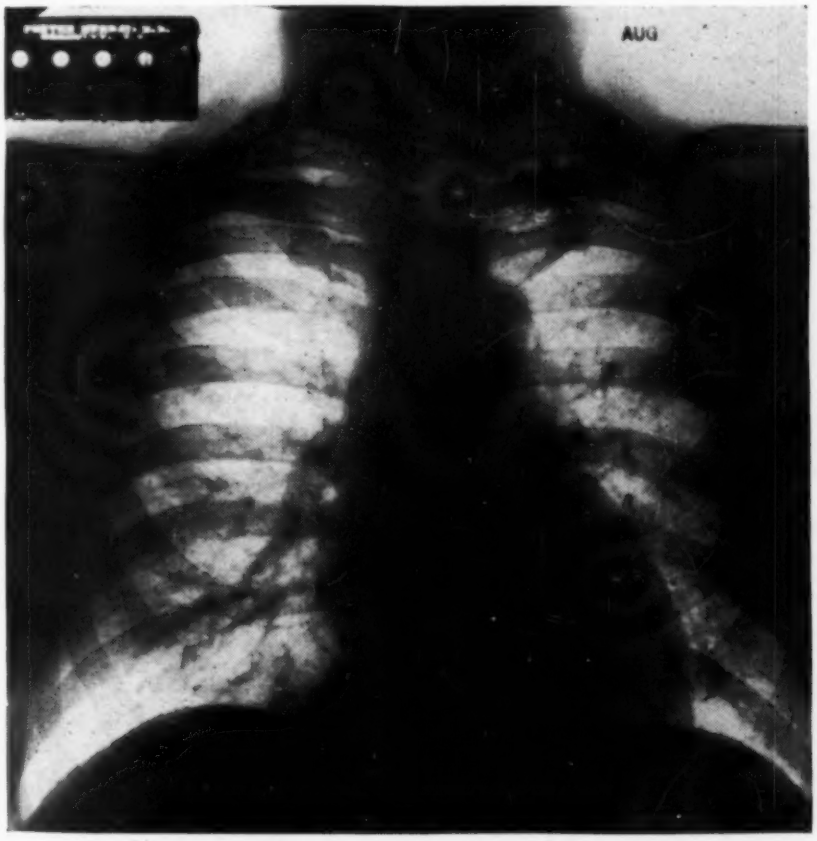

Fig. 7A—Stage in the evolution of a squamous cell carcinoma of the lung. Fairly normal left pulmonic field of a male, aged 56, taken August 3, 1936. In 1931, he had had a dense tuberculous infiltrate on this side to the fourth anterior rib. This had undergone complete resolution on a four months intensive bed rest regime with no return in the interval.

the obstruction is partial the check valve mechanism described by Chevalier Jackson[4] may operate to produce emphysema; when the obstruction is total it produces atelectasis. In either event dyspnoea follows. Massive accumulations of pleural fluid may also give rise to dyspnoea. Rarely is it toxic in origin.

4) *Wheeze*, like dyspnoea, which it frequently accompanies, caused by partial obstruction of the affected bronchus and is present in about one-third of the cases.

5) *Anorexia*, a fairly constant finding, which may arise at any time during the course of the disease.

6) *Loss of strength and weight*, a strikingly variable occurrence, frequently late in appearance.

7) *Fever*, usually a late manifestation, the result of ulceration and secondary infection. It may, however, occasionally appear early, when it rarely exceeds 100°-100.6° F.

8) *Haemoptysis*, invariably the result of ulceration and erosion,

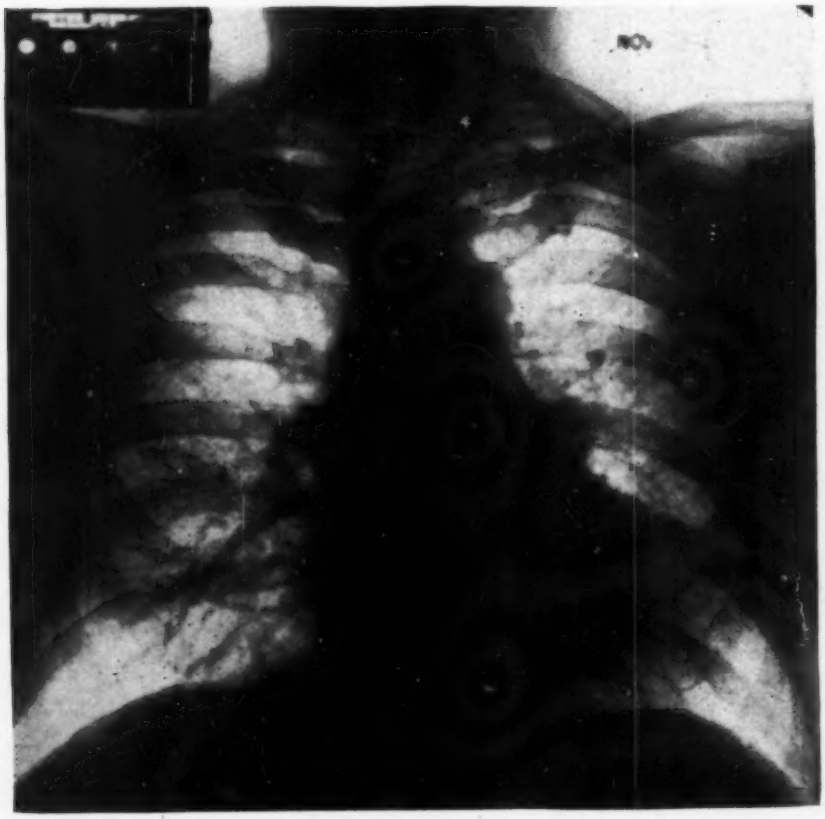

Fig. 7B—Stage in the evolution of a squamous cell carcinoma of the lung. Same case on November 14, 1938, showing small mass projecting out into left pulmonic field at about the hilum. Symptoms at this time were those of an acute upper respiratory infection of ten days duration with occasional raising of slightly bloody sputum.

occurring in a little over one-third of all cases. It is more likely to occur in the adenocarcinomatous type.

9) *Pressure symptoms*, such as dilated veins, cyanosis, edema, dysphagia, hoarseness, and elevated diaphragm, due, of course, to direct pressure of the growth on immediate adjacent structures in the mediastinal region, viz., superior vena cava, esophagus, recurrent laryngeal nerve, phrenic nerve. They are most likely to occur with the small cell type, because of its close proximity to these structures.

10) *Anemia and cachexia*, which are usually late manifestations.

11) *Secondary infection and suppuration*, the invariable result of bronchial stasis and interrupted pulmonary ventilation. These may terminate in pneumonitis, lung abscess, or bronchiectasis. They are revealed by heightened febrile response, leucocytosis, the occurrence of purulent sputum, and the development of clubbed fingers. Secondary infection and suppuration are invariably the sequelae of the more slowly evolving neoplasms; hence they occur more frequently in the squamous cell type.

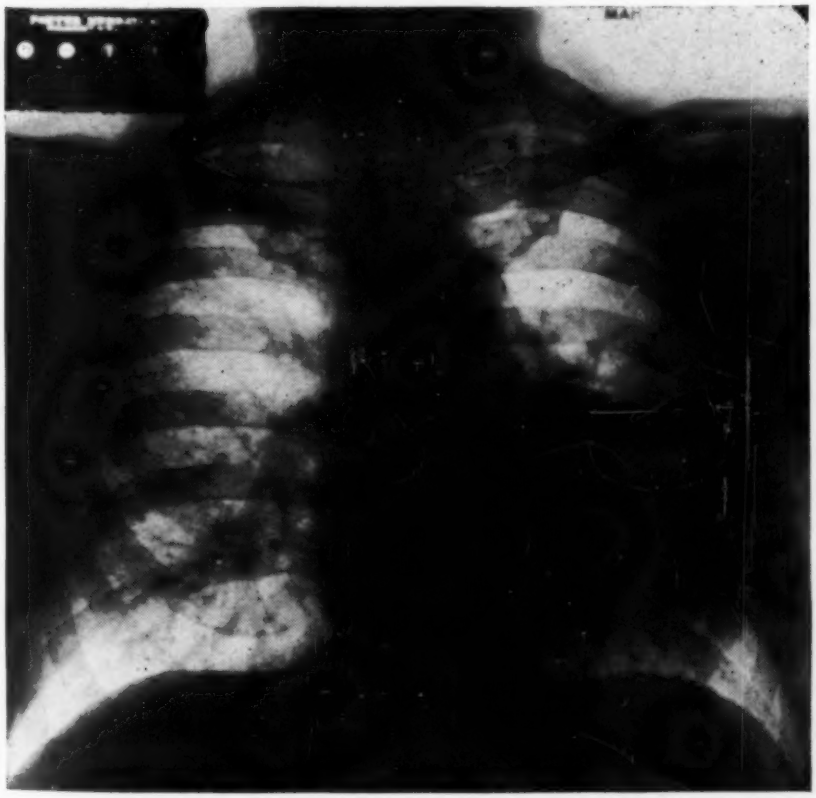

Fig. 7C—Stage in the evolution of a squamous cell carcinoma of the lung. Same case on March 17, 1939, showing development of pneumonitis distal to the mass. Cough and expectoration had slightly increased and slight dyspnoea and pain in left lower anterior chest had developed.

PLEURAL EFFUSIONS

Statistical references to the occurrence of pleural effusions vary from 15 per cent to 45 per cent. It is generally agreed they appear more regularly with the adenocarcinomatous type because of the preponderantly peripheral location of this type. About one-third of them show the presence of blood. When aspirated the fluid reforms rapidly, more rapidly than do those of other origin. Another interesting feature is that aspiration affords little or no relief from the pain which may accompany the occurrence of effusion. The fluid is of low specific gravity and low cell count. Cancer cells may be demonstrated in the centrifuged sediment. The absence of acid-fast bacilli in the sediment, determined by smear, culture, and guinea pig inoculation, serves as a differential diagnostic point.

LABORATORY FINDINGS

Sputum—There is nothing characteristic about the gross appearance of the sputum. It may run the gamut from clear mucoid expectoration to a greenish-gray, foul smelling, purulent discharge.

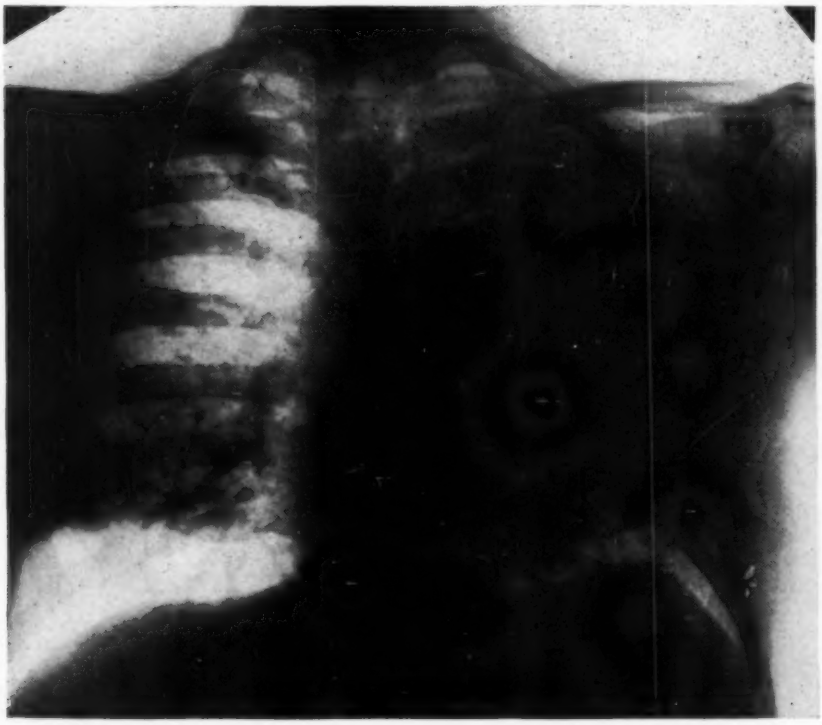

Fig. 7D—Stage in the evolution of a squamous cell carcinoma of the lung. Same case on April 11, 1939, showing atelectasis of the left upper lobe with shift of trachea and mediastinum to this side, and elevation of left diaphragm. Symptoms only very gradually increased in severity. Metastases to the left anterior cervical lymph nodes occurred in July. Death occurred October 23, 1939. Patient refused operation when advised.

The constant absence of acid-fast bacilli on repeated concentrated examination should rule out the likelihood of a tuberculous etiology. The presence of tubercle bacilli, however, does not rule out the possibility of cancer, for both may occur concomitantly.

Sedimentation Rate—The sedimentation rate is generally increased, averaging (in our series) between 20 and 30 mm. in one hour (Cutler method).

PHYSICAL FINDINGS IN CHEST

The findings on physical examination of the chest are naturally somewhat conditioned by the stage of evolution of the growth and the sequelae consequent to the mechanical and biological alterations which follow. These may vary from emphysema to atelectasis, from pneumonitis to abscess; yet the physical signs (within certain limitations) remain fairly fixed and uniform.

Invariably there is limitation of motion on the side of the chest in which the growth is located. Tactile fremitus is diminished or absent overlying the area involved and the percussion note is dull to flat. The most constant finding, however, is elicited by auscultation, and that is the *marked diminution or (more often) the absence of breath sounds over either the mass itself or the area of emphysema or atelectasis*. Rales may or may not be present. The presence of enlarged lymph nodes, particularly in the supraclavicular and axillary regions, may be noted on inspection and palpation but are of relatively infrequent occurrence.

X-RAY FINDINGS

Fluoroscopy often yields valuable information. The following are to be noted, if present:

1) Elevated diaphragm on the side of the tumor.

2) Paradoxical respiration, i.e., "see-saw" movement of the leaves of the diaphragm.

3) Swinging of the heart toward the affected side during inspiration and away on expiration.

4) Absence of expansile pulsation if the mass is located near the arch of the aorta (in contrast to aneurysm of aorta).

5) Effusion, small or massive in amount.

The *roentgenogram* is not of and by itself a decisive diagnostic measure, but when studied in combination with all other essential data gives most helpful assistance in arriving at an accurate determination. It is a highly clarifying instrument in delineating the outline, size, and especially the localization of the mass.

Primary bronchogenic carcinoma is always a unilateral entity. Evidences of contralateral involvement are due to metastases, and these are discrete, scattered, of globular configuration, and of vary-

ing size. If of small cell or squamous cell type, the parent growth usually springs from or near the hilum and arches out in semicircular contour into the body of the lung. The periphery of the mass is not too clear-cut or sharp in outline but shows strands or tongues of tissue radiating further out into the parenchyma of the lung. Yet the mass as a whole stands out, clearly silhouetted against surrounding normal lung field. Of course, when atelectasis, pneumonitis, or effusion intervene the outline is lost in the greater pervading density. If the parent tumor is of the adenocarcinomatous type, the density will usually be found further away from the hilum, out in the parenchyma of the lung, though often in the mediastinal half of the field.

Atelectasis is shown by the presence of a smooth homogeneous shadow of varying extent, depending on the number of lobules or lobes involved, together with evidence of a shift of the trachea, mediastinum, and cardiac silhouette to the same side, and a retraction of the diaphragm. Fluid, of course, casts a dense homogeneous shadow but without the evidences of shift of adjacent structures to the affected side.

Bronchography effected by use of the instillation of an opaque medium may serve more accurately to localize the atresic bronchus, but its utility is otherwise somewhat limited.

Tomography is at times distinctly helpful, not only in more clearly delineating the mass or the cavity within it, but in more definitely locating the exact position of the growth relative to the anteroposterior planes.

Roentgenography following the induction of artificial pneumothorax yields a better outline of the growth, a better appreciation of its size, and a clearer perception of complicating adhesions.

BRONCHOSCOPY

Unquestionably bronchoscopic examination adds enormously to the store of data upon which a diagnosis of bronchogenic carcinoma may be based. It is readily appreciated that only those growths which are located relatively near to the hilum fall within the vision of the bronchoscope and that many, including a large number of adenocarcinomatous type, are situated beyond that range. Hence, it must be emphasized that a negative finding does not necessarily rule out the presence of a growth.

Bronchoscopic examination may reveal fixation of the mediastinum, widening of the carina, and deformity of the bronchial walls. It makes possible direct visualization of endobronchial encroachment of the mass, all of which enriches our knowledge of the evolution of these tumors in their earlier stages and of the secondary alterations they occasion in the neighboring structures. But the

bronchoscope does more than that. It provides us with the best and safest means of obtaining a direct biopsy for histological examination. It is on such an examination alone that a *positive* diagnosis can be based. This is possible in from 70 per cent to 80 per cent of the growths lying within the range of bronchoscopic exploration. Therefore, the bronchoscope has become indispensable in the diagnosis of bronchogenic carcinoma. One should not fail to insist on its employment the moment a presumptive diagnosis, based on a study of all previous data, is made.

OTHER AIDS TO DIAGNOSIS

Biopsy material may be obtained for histological examination when superficial lymph nodes give evidence of adenopathy.

When x-ray densities are peripherally located and found to be beyond range of the bronchoscope, direct transthoracic puncture may be resorted to and tissue aspirated for histological examination. Many clinicians warn against this practice, but there are clinics in which it is employed with apparent impunity.

Again, with the more peripheral tumors artificial pneumothorax may be performed, followed by a thoracoscopic exploration and, where found feasible, a biopsy section obtained.

When other methods of obtaining biopsy material fail an exploratory thoracotomy is justified.

CAUSES OF DEATH

The causes of death in bronchogenic carcinoma are many and varied. They are conditioned largely by the type and grade of malignancy of the tumor. More deaths are probably caused by the complications or sequelae of the disease than by the growth itself. Among these, secondary infections and metastases play a major role, particularly metastases to the brain and other vital organs. Pneumonia, edema of the lung, lung abscess, and bronchiectasis occur more frequently with the squamous cell type. Asphyxia and cachexia may occur with any type but predominate with the small cell group. Hemorrhage (sometimes massive) and cardiac decompensation occasionally occur in all forms, but slightly more with the adenocarcinoma group. It is with the latter group that metastases to the brain, liver, contralateral lung, and long bones seem especially prone to occur.

PROGNOSIS AND TREATMENT

Without treatment the prognosis is fatal.

Surgery offers the only possible chance of cure. Despite the encouraging reports of radio-therapeutics by Leddy and Moersch,[7] the consensus is that nothing short of prompt and complete extir-

pation of the growth offers any reasonable prospect of lasting result. Even the success of surgical attack is severely circumscribed by such factors as the time element, the type and grade of malignancy, the location of the growth, the occurrence of metastases, and pleural effusion; and last but not least the general physical condition of the patient. Most of the small cell group are inoperable by the time the diagnosis is established. This is because they so often arise from the major stem bronchus, or very close thereto, and so early and vigorously invade the mediastinal lymph nodes. With the adenocarcinomatous group, remote (hematogenous) metastases frequently occur before symptoms directing attention to the chest arise. Hence, by the time the parent growth in the lung is recognized it is obviously too late for surgical attack. About the only group which offers any measure of success in surgical attack is the squamous cell. This comprises about 42 per cent of all such tumors. The operations of choice are lobectomy or pneumonectomy, depending chiefly on the location of the growth. Churchill,[8] Overholt,[9] and Rienhoff[10] have given excellent descriptions of these operations.

CONTRAINDICATIONS FOR SURGERY

Surgery is contraindicated in the presence of:

1) Evidence of metastases, in mediastinum or peripheral lymph nodes, or in brain, bone, liver, or other more remote structures.

2) Pressure symptoms—dilations of superficial veins of chest, edema, dysphonia, diaphragmatic paralysis, dysphagia, etc.

3) Pleural effusion, with rare exceptions.

CONCLUSIONS

The incidence of bronchogenic carcinoma has definitely increased. There should be a much greater awareness of this fact. There should be a much greater alertness in recognizing its occurrence in its earliest stages.

It occurs most frequently in the age bracket of 40-60 years. Cases whose symptomatology simulates that of clinical pulmonary tuberculosis, asthma, or pleurisy, in that age period, should make one suspect the possibility of malignancy. Beware of the cough, the wheeze, the haemoptysis, and the pain in the chest that start and endure after 40 years of age, especially if such symptoms have not occurred before.

When tumors are discovered in the brain, the kidneys, the adrenals, or the long bones, attention should be directed to the lungs. Roentgenographic and physical examination of the chest may often reveal the presence of the parent growth in the lung.

The most constant findings on physical examination of the chest are limitation of motion, dull to flat percussion note, and especially

absence of breath sounds on one side of the chest or a portion thereof.

The roentgenogram contributes most useful confirmatory evidence. It helps more clearly in outlining the mass and in defining its size and position, especially if supplemented by tomographic serials, bronchography, or artificial pneumothorax.

When bronchogenic carcinoma is suspected but not proved, bronchoscopic examination should be considered mandatory. By means of the bronchoscope biopsy material may be obtained for histological examination. Only by such an examination may a positive diagnosis of malignancy be established.

The prognosis in untreated cases is fatal.

Radiotherapy is at the best but palliative.

Radical surgery—lobectomy or pneumonectomy—offers the only prospect of cure. To be effective, operative measures must be employed at the earliest possible time, before metastases or other fatal complications occur; hence, the vital importance of *early diagnosis*.

CONCLUSIONES

Ha aumentado definitivamente la frecuencia del carcinoma broncógeno. Debe tenerse este hecho mucho más en cuenta. Debe existir mucha más diligencia en reconocer la aparición de este mal en sus períodos más tempranos.

Aparece más frecuentemente en el período etario de 40 a 60 años. Aquellos casos en este período etario cuya sintomatología simula la de la tuberculosis pulmonar clínica, el asma o la pleuresía, deben despertar en uno la sospecha de la posible existencia de la malignidad.

Sospéchese de la tos, de la respiración ruda, de la hemoptisis y del dolor en el pecho que comienzan después de los 40 años de edad y que continúan, especialmente si estos síntomas no han existido antes.

El descubrimiento de tumores en el cerebro, los riñones, las cápsulas suprarrenales o los huesos largos, debe fijar la atención en los pulmones. El examen físico y roentgenográfico del pecho a menudo revela la presencia de la neoplasia primaria en el pulmón.

Los hallazgos más frecuentes del examen físico del pecho son: la limitación del movimiento, una nota de submate a mate en la percusión y, especialmente, la ausencia de los ruidos respiratorios de parte o de todo un lado del pecho.

La roentgenografía suministra evidencia confirmatoria sumamente útil ya que ayuda a delinear el tumor más claramente y a determinar su tamaño y posición, especialmente si se le suple con tomografías, con la broncografía o el neumotórax artificial.

El examen broncoscópico debe considerarse obligatorio cuando se

sospecha, pero no se ha comprobado, la presencia del carcinoma broncógeno. Por medio del broncoscopio puede hacerse una biopsia para obtener tejido para el estudio histológico. Sólo mediante tal examen es posible establecer el diagnóstico positivo de la malignidad.

Sin tratamiento el pronóstico es fatal en todos los casos.

La radioterapia es cuando más solamente paliativa.

La cirugía radical—lobectomía o neumonectomía—ofrece la única esperanza de curación. Para ser eficaces, las medidas operatorias deben emplearse lo más temprano posible, antes de que sobrevengan metastasias u otras complicaciones fatales; de aquí la importancia vital del diagnóstico temprano.

80 Hanson Place.

REFERENCES

1 Perrone, J. A., and Levinson, J. P.: "Primary Carcinoma of the Lung," *Annals of Int. Med.*, 17: 12, (July) 1942.

2 Menne, F. R., and Anderson, M. W.: "Bronchogenic Carcinoma," *J. A. M. A.*, 117: 2215, (Dec. 27) 1941.

3 Ewing, James: *Neoplastic Diseases;* Fourth Edition, p. 878, W. B. Saunders Co., 1940.

4 Jackson, Chevalier: "The Mechanism of Physical Signs in Neoplastic and Other Diseases of the Lung," *J. A. M. A.*, 95: 639, (Aug. 30) 1930.

5 Gebauer, Paul W.: "The Differentiation of Bronchogenic Carcinomas," *Jour. of Thoracic Surgery*, 10: 4, (April) 1941.

6 Simons, Edwin J.: *Primary Carcinoma of the Lungs*, The Year Book Publishers, Inc., Chicago, 1937.

7 Leddy, Eugene T., and Moersch, Herman J.: "Roentgen Therapy for Bronchogenic Carcinoma," *J. A. M. A.*, 115: 2239, (Dec. 28) 1940.

8 Churchill, Edward D.: "Resection of the Lung," *Surgery*, 8: 961, (Dec.) 1940.

9 Overholt, Richard H.: "Pneumonectomy for Malignant and Suppurative Disease of the Lung," *Jour. of Thoracic Surgery*, 9: 17, (Oct.) 1939.

10 Rienhoff, William Francis, Jr.: "Graded Pneumonectomy in the Treatment of Tumors of the Lung," *Bulletin of the Johns Hopkins Hospital*, 64: 167, (March) 1939.

Tuberculosis

Weather and Resistance*

WILLIAM F. PETERSEN, M.D.
Chicago, Illinois

An interest in the atmosphere in which we have our being might seem particularly appropriate for the physician concerned with the organs of respiration. But, as is often the case, the commonplace is so common that we ignore it, the while we cultivate the exotic.

A consideration of this mental blind spot is to be found in Lawrence Henderson's[1] essay, "The Study of Man," from which the following paragraph has been taken:

"The disposition to think in this manner (i.e., to regard a phenomenon as simple, its cause as single and to feel that accordingly there is but one clear, straight path to be followed in action) has been strengthened both by the conditions of modern life upon the practice of medicine and by increasing specialization. So it comes about that sick people are often regarded as cases rather than persons, and many things in the history of their sickness, many others concerning their environment, which were familiar to general practitioners of an earlier day, are likely to be overlooked and not be suspected. And yet, disregard of such factors or others that seem unimportant in the light of oversimplified theory often leads to disaster. Our disregarded factors, as often as not are among those which the old-fashioned practitioner intuitively recognized and which can sometimes be discovered only through intimate acquaintance with the patient and familiarity with his environment, with his life day by day, and with his family history."

This point of view, so ably developed by Henderson, might well preface the material that I shall discuss, for it deals with one of these disregarded environmental factors, namely, the weather. So thoroughly disregarded has it been for the past half century that even the mention of the topic is met with raised eyebrows and shrugging shoulders.

Today man seems so far removed from the environment; he is presumably so well sheltered and clothed that change in the air that he breathes, that penetrates every part of his body and bombards every inch of his surface is regarded as quite unimportant; environment today, to the physician, concerns the social milieu, the schooling, the occupation, the domestic situation—never so commonplace a thing as weather.

---

*Presented at the Annual Meeting of the American College of Chest Physicians, Atlantic City, New Jersey, June 7, 1942.

I would immediately counter with the seemingly categorical statement that the human organism is wholly weather conditioned, that the day-by-day change in the air mass that we breathe, the seasons, the climatic cycle, are of great significance in conditioning the reaction of the normal, and of major significance for the sick individual—and always will be. As long as we are healthy, the organic integration with weather is so perfect that we are wholly unaware of the association; only when some organ or tissue becomes inadequate does the clinical record reveal the effect and, the sicker the patient, the greater the effectiveness.

Under the circumstances, it might seem a legitimate excursion for the physician dealing with respiratory diseases to examine some of these forces and to become aware of their significance.

To resolve our problem into simple terms we can assume two weather (air mass) states; one which causes the body to lose heat (cold, increased wind velocity, increased moisture content at certain temperatures, less sunshine) and one which makes it difficult for the body to lose heat (high temperatures, high humidity, less wind circulation, more sunshine, etc.).

With the first, the body mechanisms will seek to increase its insulating power, with the latter its radiation, i.e., opposite physiological states. The former is accomplished primarily by peripheral vasoconstriction, the latter by means of peripheral dilatation. To accomplish this, an elaborate nervous, chemical and endocrine integration becomes effective. These mechanisms that control the maintenance of our body temperature have reached a particularly high degree of development in the hairless human. Heat regulation, with its use of vascular control for radiation or insulation, may at times interfere with and proceed at the expense of proper oxygenation of tissues, which is, after all, more important than heat regulation. It is this basic conflict of most imperative functions that leads not infrequently to the patient's discomfort or disease, which we study in objective symptomatology.

In the presence of an infection such as tuberculosis, we can observe this resulting dysfunction of the organic balance in changing tides of increasing or decreasing resistance; in the asthmatic, in increased or decreased discomfort; in the patient with pulmonary malignancy, with greater or lesser tendency to dyspnea, to hemorrhage, to general well being or distress.

Weather effects might present a simple field of study were it not for reasons already stressed by Henderson, namely, that the effects are never single nor simple.

In the first place weather change is a complex change. The air mass, or aerosol, is a restless medium and involves the interplay of many physical forces: temperature, moisture content, friction (wind

velocity), cooling power associated with air movement, transmission of radiant energy, variation in ionization of the molecule, in the pressure and in the oxygen content as well—all these being effective on a variety of organic receptors such as skin, the mucous membranes and lungs, the special sense organs, etc.

With this, we must keep in mind that no weather effect is ever repeated—an important consideration. From the purely theoretical point of view the receptor (the human) is always older and thus the reactivity changes; from the practical standpoint the organism is always changed and conditioned by the *preceding* weather change and the sequence of weather change is never identical.

We must keep before us the recognition that the mechanisms of adjustment initiated by weather change (vascular, chemical, endocrine) are the same mechanisms involved to meet other environmental impacts whether traumatic, infectious, due to work, to emotional disturbances, etc.; these various effectors set up waves of increasing or decreasing organic functions which in turn may either be negated or augmented by succeeding impacts and inherent organic rhythms (diurnal, sex, seasonal, etc.).

For those of us living in America a knowledge of the circumpolar cyclonic circulation of the atmosphere is of particular importance because the majority of us live in the pathway of the mixing zone of polar and tropical air masses, with constant change from heat to cold or cold to heat.*

When a polar air mass (the anticyclone, rotating clockwise and relatively stable) passes over us, we breathe air that contains more oxygen, is denser, heavier (the barometric pressure increases), drier, colder and clearer. In general we increase our insulation, the peripheral vessels contract, blood pressure increases, the majority of normal individuals experience a sense of well being.

When a tropical air mass passes (i.e., the cyclone rotating counter-clockwise and progressing rapidly) we breathe air that is diametrically opposite in its physical properties.

These air masses are clearly definable and at the interface or plane of contact (fronts) of air masses of such totally different character, precipitation phenomena occur with the characteristic symptoms that we recognize as storms (with fog, rain, snow, violent winds, etc.).

We can illustrate the blood pressor reaction that is associated with the passage of a cold front and divide the reaction into two phases.

---

*Many readily available popular, as well as scientific monographs dealing with weather have appeared in recent years. It would lead too far afield to discuss details of meteorology in this paper

First, the phase of increased insulation and resistance when:

1) Vessels contract ("adrenal" phase).

2) Local anabolism, with lessening of the oxygen supply; reduction phenomena may dominate.

3) Less CO_2 is produced.

4) Blood coming from the area is relatively more alkaline (the while the tissues may become more acid).

Second, the compensatory phase which now follows:

1) Vessels dilate, there may be (a) active hyperemia with capillary dilatation, increased capillary permeability, hydration of the tissues, etc., ("thyroid" phase); (b) venous stasis.

2) Catabolism is accelerated, oxidation is enhanced.

3) More CO_2 is produced.

4) The blood coming from the tissues becomes more acid (increased CO_2, lactic acid, etc.).

This is a normal reaction to environmental impacts; it recurs constantly, with modification of rhythm and amplitude by interference phenomena. Obviously amplification, such as illustrated in the 2nd and 3rd curves (Fig. 1), may lead to clinical episodes (here labeled hemorrhage with pressor periods, and thrombosis when stasis may follow and pressures are low).

These reactions occur whether the patient is in bed and in an enclosed room or is up and about; obviously shelter and rest will modify the reactivity.

This can be illustrated if we merely follow the day-by-day blood pressure curve of an old woman observed over a two-year period. She had a senile sclerosis and the pressures were high, but the vast crests and depressions are due to weather change.

In this curve (Fig. 2) characteristic seasonal trends can be ob-

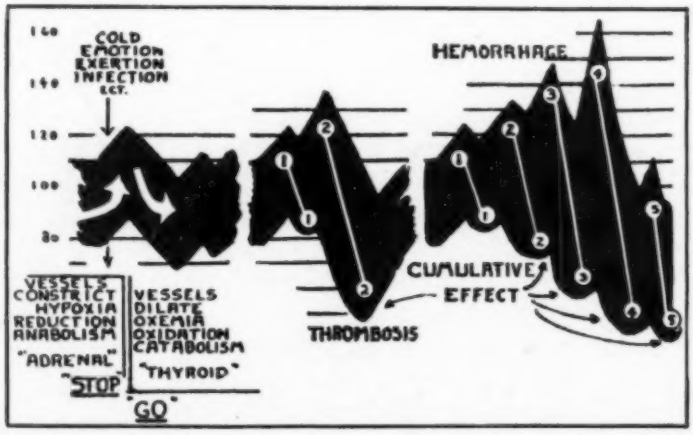

Fig. 1—Diagram to illustrate single, double, and repeated environmental impacts on the organism, with diphasic vascular response as well as summation phenomena with the appearance of clinical episodes

served: in the midsummer (S) the pressure levels declined because of peripheral dilatation. In the autumn (with December crest) pressure levels again were high (A), in part due to the increase in buffer and relative alkalinity, and in part because of declining temperature. After these pressure levels again reached a maximum, they declined rather suddenly in January (J). But this was not due to dilatation because of peripheral warmth, this time the decline in blood pressure was due to the production of greater quantities of capillary-active substances when the vascular bed was spastic (pressor crests) and a relative anoxia prevailed. This decline was due to vascular fatigue.

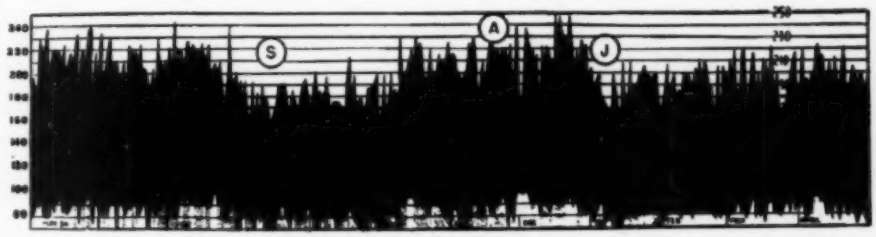

Fig. 2—Daily systolic and diastolic blood pressure readings on subject M., May 1934 to May 1935

In Figure 2A the first two months of the pressor curve is presented on a larger scale with a superimposed meteorogram. On the meteorogram wind velocity, mean daily temperatures, and barometric pressure is indicated. Blood pressure episodes 1-9 have been indicated over the blood pressure curve and corresponding numbers on the meteorogram indicate the associated periods of decreasing environmental temperature and increasing barometric pressure. It is to be understood the patient was under uniform environmental conditions (food, shelter, emotions, etc.). Despite this it will be observed that every change in the air mass was reflected in a change in the blood pressure levels and with this there is, of course, a corresponding change in practically every biological index.

Most physicians have accepted the inclusive and very satisfying term *homeostasis*, with its assumption that the "humours," to use the old Hippocratic term, are kept constant in composition by the very elaborate machinery evolved for just this purpose. They are, within limits.

"All other things being equal" has been the accepted formula of the physiologist and biochemist. But in this most turbulent of worlds all things are never equal. Lawrence Henderson took evident delight in making clear the significance of this fact and in my own work I have become almost painfully aware of the constant change in the organism and its external milieu.

To illustrate the range of such a presumably stable indicator as the pH of the venous blood of the arm, the next set of curves (Fig. 3) will be presented. Here the day-by-day pH determinations, made each morning for a six-week period under identical conditions on three young adults (triplets) are portrayed and over the curve the meteorogram has been carried to illustrate the weather of the time.

The correlation coefficients of the pH values are all significant, i.e., these, like individuals, reacted in a similar fashion; they reacted to the weather. With each passage of a cold air mass, an alkalotic crest was reached; then followed a period of relative

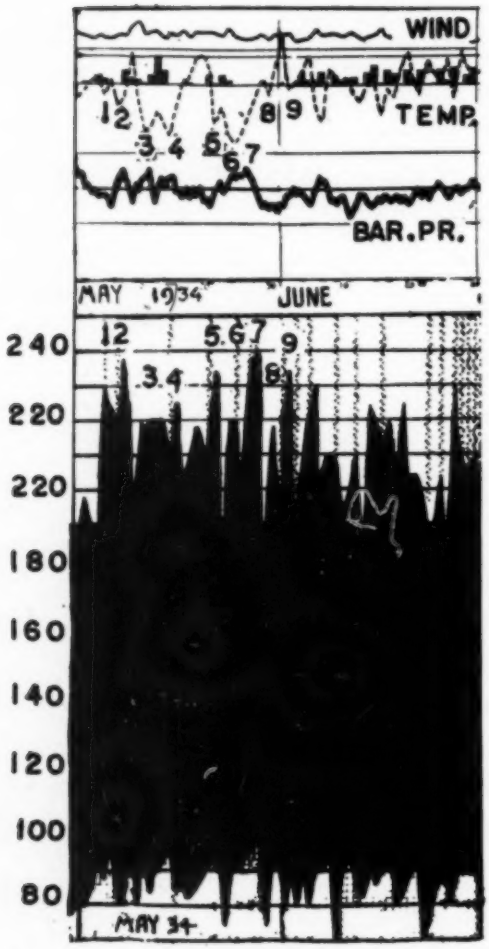

Fig. 2A—The blood pressure curve for the months of May and June, 1934, with superimposed meteorogram. Pressor episodes during May have been numbered 1-9.

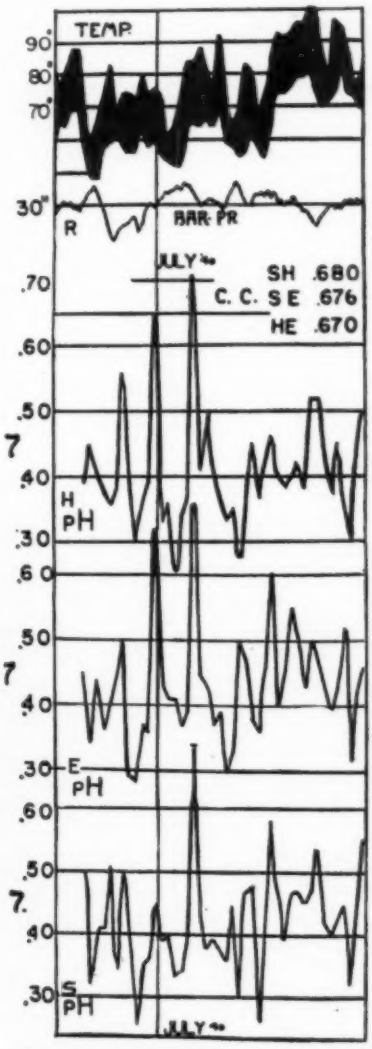

Fig. 3—Curves of day-by-day pH determinations of venous blood of the arm on a set of adult triplets observed during June and July 1940.

acidity as a corrective swing. When extreme temperatures (102" on July 25) were reached, the pH was again high—this time for a wholly different reason—in part because of the more rapid elimination of CO_2 by increased ventilation, in part because of the loss of chlorides at the periphery.

VITAL INDEX

Instead of dealing with like individuals (a preliminary report on some of this material has been published[2]), we turn to a survey of the entire population during this six-week period. We determined a three-day moving average of the daily deaths in Chicago and a three-day moving average of the conceptions for the same period (birth 280 days later). The conception/death ratio gives us a vitality index of the biological status of the population at large.

When we examine this curve (Fig. 4), it clearly reveals a continual pendulation, with a crest on June 26 (near the summer solstice) and a low a month later. A glance at the superimposed meteorogram makes it evident that at this time of the year each passing cold air mass apparently increased the vitality.

When we now examine this curve of biological vitality as it is related to the pH curve of the triplet group already discussed, a relation is obvious. The chemical shift in our normal triplets, weather conditioned, is related to the vitality index of the population at large, i.e., the normal population reflects the individual reaction,

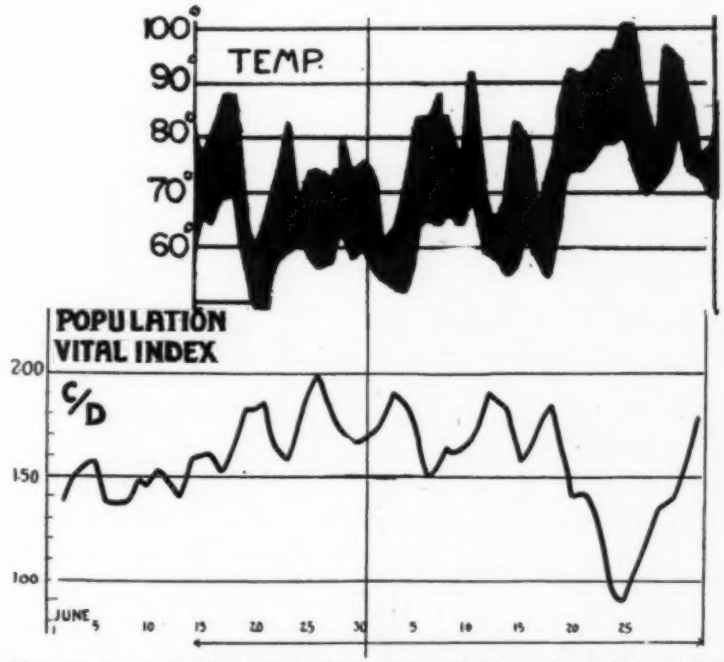

Fig. 4—The curve of population vital index (conception/death) for the period of June and July, 1940.

the whole population moves in biological accord with the meteorological environment. Thus observations that we make in one individual or in a group are related to biological events occurring in other individuals observed under similar conditions at the same time.

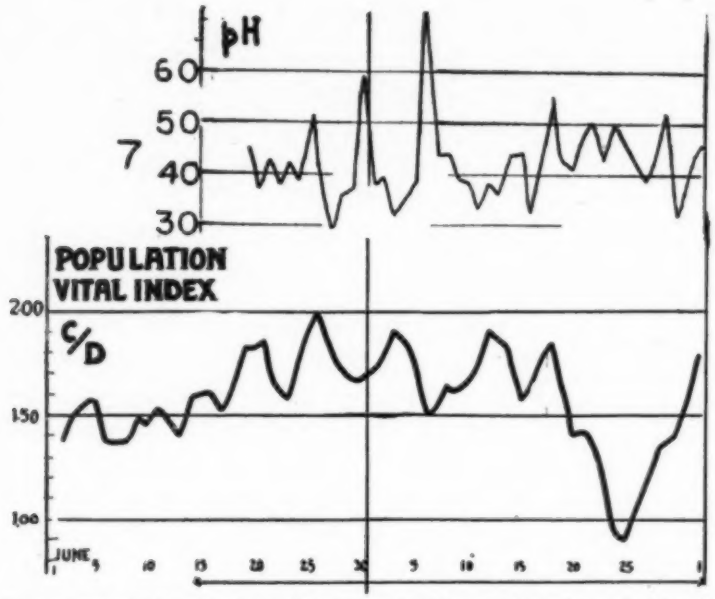

Fig. 5—The mean pH levels of triplet group for the period of June 19 to the end of July, 1940, superimposed over the curve of population vital index.

If the reader will now accept the fact[3] that every biological balance (whether hydration or dehydration, cholesterol-lecithin, calcium-potassium, hypo- or hyperglycemia, lymphocytic or leucocytic, trypsin-antitrypsin) is swinging in a corresponding phase, and will apply this concept of a labile rhythm to the milieu of the tubercle, the immediate clinical significance will become apparent:

1) A quiescent tubercle heretofore in balance with its humoral and cellular milieu may proceed toward *healing*. This will in general involve (a) a state with connective tissue overbalance, (b) with greater tissue cohesion and less permeability, (c) lessened accumulation of acids, (d) the trend to the deposit of calcium and collagen, (e) lessened digestive phenomena, (f) lessened diffusion of tuberculous and non-specific products of tissue digestion, (g) lessened growth of bacilli, (h) lessened general organic fatigue, together with many related changes. Anabolism and reductive phenomena dominate.

2) A quiescent tubercle, heretofore in balance with its humoral and cellular milieu, may proceed toward *activation*. This will involve (a) lessened cohesion and greater hydration, (b) poorer connective

tissue reaction and lessened deposit of calcium as well as connective tissue fibrillae, (c) greater accumulation of acids with (d) greater local digestive activity, greater diffusion of tuberculous and non-specific capillary-active substances, (e) growth and dissemination of bacteria, greater general fatigue phenomena, etc. Catabolic and oxidative phenomena dominate.

All this, of course, leaves out of consideration the specific factors which are also involved.

Any number of environmental impacts can swing this humoral milieu. It will be affected by food and emotion, by trauma and work, by infections and intoxication—all on the background of the constitution of the individual, the age, the sex, the previous exposure, etc. But in the forefront must be considered the weather of the time, the season (accumulation of weather effects), and the phase of the climatic cycle (i.e., the amplification of seasonal effects) in which the individual happens to exist.

THE SIGNIFICANCE OF THE DIPHASIC CHARACTER OF ORGANIC REACTIONS

Every energy impact, whether weather, trauma or infection, is followed by a diphasic reaction in biochemical levels and for purposes of orientation, we can express these best in terms of the acid-base balance (pH levels) and is illustrated in Figure 6.

In this diagram we can assume that in the late winter and spring an energy impact involving a tuberculous patient (and the tubercle) at a time when the pH levels of the venous blood of the arm are at approximately 7.40, will result first in an increase (A) to 7.50, then shortly there will follow a swing to the acid side, reaching levels of 7.30 a day or so later (B).

These impacts are repeated. Finally a point is reached after five successive impacts when (with exhaustion of reserves) a level of 7.20 is reached. Here catabolic (digestive and disintegrative) phenomena may be accelerated to such an extent that activation occurs.

Spring Activation—This is the underlying cause of the characteristic spring activation of the tuberculous individual with which we are all familiar. A pH curve made up of several thousand observations on normal as well as sick individuals during the decade 1930 to 1940 reveals the characteristic spring lowering of pH levels, reaching their nadir in April. This coincides with the period of least resistance to tuberculous infection (Fig. 7).

Autumnal Quiescence—Summer and autumnal quiescence is almost as apparent in the tuberculous history as is spring activation and the diagrammatic text figure (Fig. 8) will serve to illustrate the mechanisms involved, being the converse of the spring effect.

Obviously the clinical course, the tide of increasing or decreasing

resistance does not follow a smooth curve;[4] it is made up of a multitude of major and minor reactions which determine the outcome, both as far as general trends are concerned, as well as the violent individual crises that follow environmental changes.

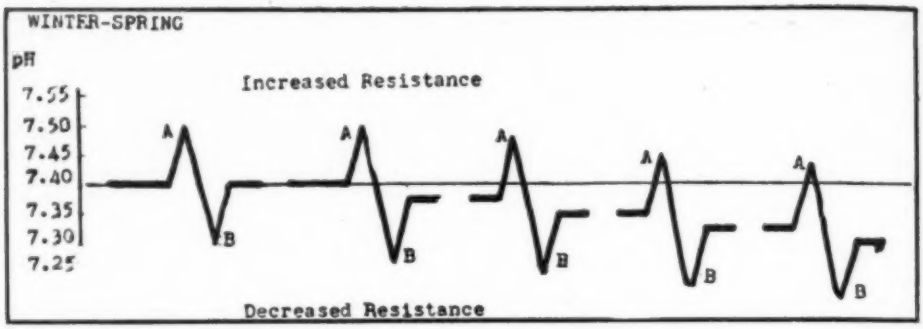

Fig. 6—Acid-base balance (pH levels) changes to illustrate the diphasic reaction to energy impacts in the late winter and spring.

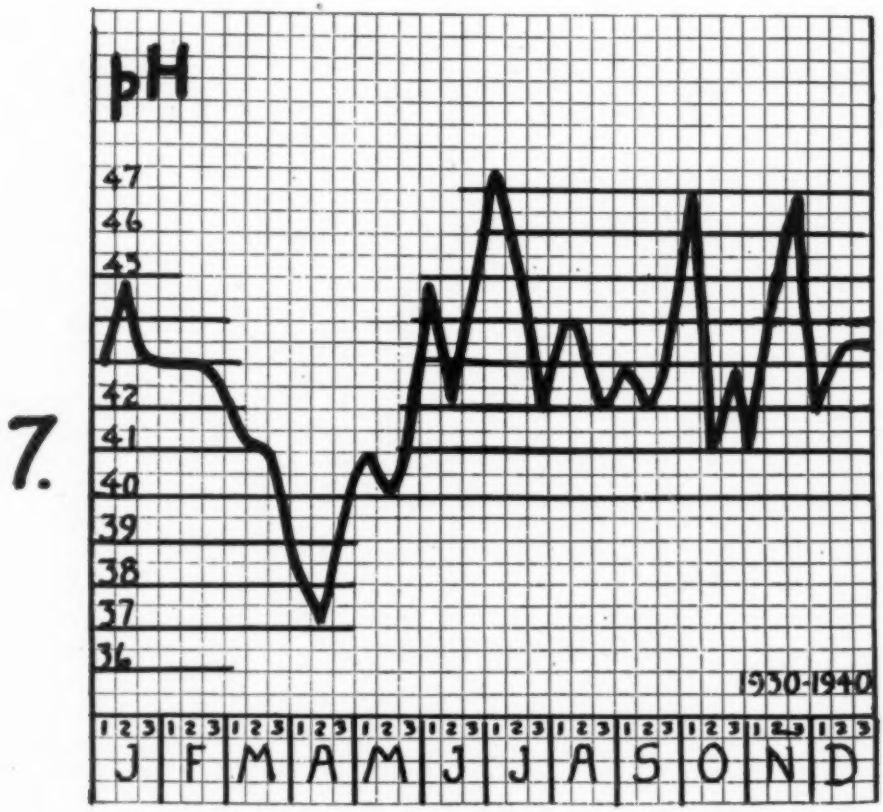

Fig. 7—Curve of pH levels of the venous blood of the arm, made in day-by-day fashion for the period 1930-1940. Note the characteristic lowering of level to its April decline to relative acidity and the sharp increase in summer and autumn.

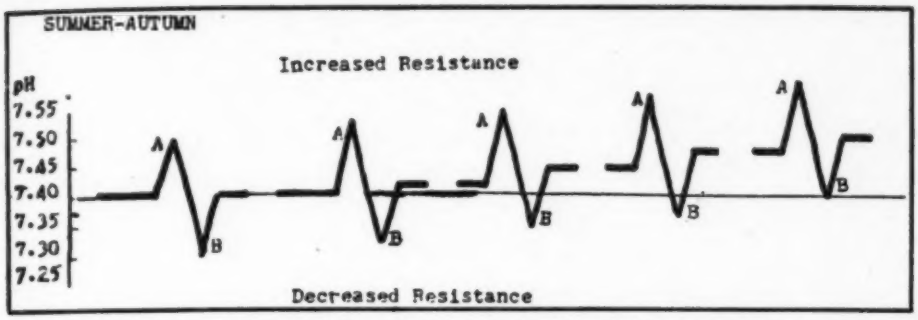

Fig. 8—Acid-base balance changes to illustrate the diphasic reaction to energy impacts in summer and autumn.

THE CLINICAL EPISODE

Through the efforts of my associates (Dr. J. S. Howe, Margaret Milliken, Mrs. F. C. Whitcomb), both experimental as well as detailed clinical material[5] has been published to provide the background of validity for this thesis. Rather than select clinical episodes from a region such as ours, where the association of weather and the precipitation of clinical symptomatology is rather clear-cut because of the brusqueness of the impact, I shall illustrate the concept as it is reflected in the clinical course of a patient in regions where the environment is less taxing and where, as a consequence, the association of clinical changes with weather change is less easily followed. I make use of a report in the literature, "Tuberculous Mediastinitis," by R. H. Kampmeier.[6] The history is as follows:

A young man, 18 years of age, was admitted to Charity Hospital in New Orleans on March 8, 1935 (4) (Fig. 9). His chief complaint was shortness of breath, cough, and pain and swelling of the left arm. He became aware of weakness in January 1935 and was told at that time that he was pale; he felt tired much of the time. At about the same time a dull aching pain developed in the left chest, radiating to the lower costal margin of that side. About a week after the onset of pain the patient began to be troubled with a sore throat and a productive cough. From the onset night-sweats were profuse; chilliness was often noted in the daytime. We have no exact dating for this onset other than that it was in January.

Three weeks before admission dyspnoea was noted on exertion (1) and this symptom has continued. He consulted a physician because of this symptom on February 21 (2). A diagnosis of pleurisy was made and a pint of yellow fluid was removed from the left chest. Three days after the aspiration (3) the patient developed pain in the left shoulder. One and a half weeks before admission he awoke one morning to find his left arm swollen from shoulder to hand. Two weeks before admission he began to have diarrhoea. The stool was of small quantity, tenesmus was present and the diarrhoea lasted one week. The first x-ray examination on March 11 was reported as showing dry mediastinitis and pericarditis.

Without going into details of the physical examination, the progress notes indicate that ten days after admission (5), the edema of the left

arm had begun to subside, and the patient was complaining of pain in the right arm and of tenderness in the right axilla. On the following day, March 19, the right arm had swollen, being more edematous than the gradually improving left arm. Four days later the swelling of the right arm had become so marked that the radial pulse could not be felt and it was impossible to obtain a blood pressure reading. The patient continued to complain of varying symptoms. During the month of May and the first half of June the patient was at his best. He had no edema of any part, only slight cough and no dysphagia. Much of the time was spent out of bed. He ate quite well. No fever was present at any time.

At about the middle of June (6) the patient developed fever for the first time after admission. On the 15th of June some clear yellow fluid was aspirated from the right chest, as had previously been done on May 20 and 27. The fever which began at this time lasted until the time when he left the hospital; at this time, too, the patient went to bed because of abdominal pain. This was diffuse, quite severe, and with little muscle reaction. Diarrhoea developed about ten days after this and the patient began to lose strength and weight. The patient was discharged against advice, upon the insistence of his family, on July 6 and died on July 21 (7).

A meteorogram has been prepared for the weather of New Orleans. In this, the daily maximal and minimal temperatures are indicated in the black field, the barometric pressure by the solid line, and precipitation by the black columns below the date line.

The dates of the clinical episodes described are indicated by circled numbers.

The onset of active symptomatology in January and February occurred in a period of considerable environmental variation. In the south, chilling may at times be severe because of the relative moisture content of the air as well as because of poor housing and clothing.

The first dating of a clinical change is only approximate (i.e., three weeks before admission, when (1) he noted dyspnea on exertion).

This corresponds to a time of low barometric pressure and high temperature (passage of a cyclonic air mass). Blood pressures would have been lower, hydration and diffusion of toxin greater, and permeability of capillaries increased. The increasing permeability of the vascular bed had become evident in a pleurisy.

Increase in blood pressure and the vasoconstriction associated with the colder air mass that now passed (note barometric pressure increase) caused greater distress and the patient first consulted a physician; at this time fluid was withdrawn from the chest (2).

With the next period of rain, the patient developed pain in the shoulder (3). Admission to the hospital (4) occurred at a time of great precipitation (4"), with sharp fall in temperature and an increase in barometric pressure. Obviously the accentuation of illness

which finally forced the patient to the hospital was associated with this violent environmental disturbance.

The succeeding period of severe rain (March 17) was again associated with a clinical change—this time with pain in the right arm and axilla and swelling of the right arm (5).

With increasing environmental temperatures and relative environmental stability, the patient temporarily improved, but about the middle of June progression was again evident (intestinal and peritoneal) with higher temperatures (6). Death (7) occurred shortly after a barometric crest, when environmental temperatures had declined from a high of approximately 100° F. and when precipitation was again apparent after the 17th of July. *See reference 7.*

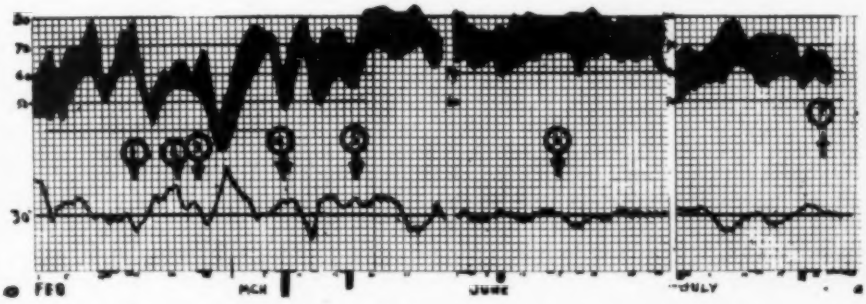

Fig. 9—Meteorogram to illustrate clinical events in a case of Dr. Kampmeier.

DISCUSSION

Weather must not be regarded an unusual or mysterious effector for normal or sick individuals. The pathophysiological mechanisms involved are the same that are concerned with reaction to trauma (emotional or physical), infection, intoxication, work or fatigue, or endocrine rhythm. In the population the sick, as well as the very young or the very old, become more susceptible than the normal; the active tuberculous patient is more susceptible than the individual who has a quiescent or healing lesion—but in all cases the biochemical tide involved, initiated by weather and season, is one that must be considered by every physician. The reaction to environmental change is a basic one. It may aid or harm the patient in overcoming the infection. The direction and degree can only be evaluated by close observation of the patient by the clinician aware of the possibilities for good or evil.

This is a point of view that has repeatedly been stressed by Pottenger[9] and by Mills.[10] They have called attention to the weather as one of the essential factors that must be kept in mind when we consider the factors that have to do with resistance to tuberculosis.

Considerations of this kind are of practical significance in problems of surgical intervention, of residence, as well as of rehabilitation and exercise.

DISCUSION

No debe considerarse al tiempo una causa extraña o misteriosa en la condición de sujetos normales o enfermos. Los mecanismos patofisiológicos implicados son los mismos que tienen que ver con la reacción al traumatismo (emocional o físico), a la infección, a la intoxicación, al trabajo o la fatiga, o a la periodicidad endocrinal. En la población en general los enfermos, así como los muy tiernos y los muy viejos, se vuelven más susceptibles que los normales; el paciente de tuberculosis activa es más susceptible que el individuo que tiene una lesión inactiva o cicatrizante; pero en todo caso la corriente bioquímica implicada, iniciada por el tiempo y la estación, debe ser tenida en cuenta por todo médico. La reacción al cambio del ambiente es fundamental y puede ayudar o perjudicar al paciente en su lucha contra la infección. La dirección y el grado sólo pueden avaluarse mediante la observación íntima del paciente por el clínico bien enterado de las posibilidades que existen para la producción de bien o mal.

Este es un punto de vista en el que Pottenger[9] y Mills[10] han hecho hincapié repetidamente. Ellos han llamado la atención al hecho de que el tiempo es uno de los factores esenciales que deben tenerse en cuenta cuando se consideran los factores que tienen que ver con la resistencia a la tuberculosis.

Reflexiones de esta naturaleza tienen un significado práctico en resolver problemas de la intervención quirúrgica o del lugar de residencia, así como de la rehabilitación y del ejercicio.

1322 Astor Street.

REFERENCES

1 Henderson, L. J.: "The Study of Man," *Science*, 95: 1, 1941.

2 Petersen, W. F.: "Weather and Biochemical Variability," *Arch. Biochem.*, 1: 269, 1942.

3 Peterson, W. F., and Milliken, M. E.: *The Patient and the Weather*, Vol. IV, Part 2; *Organic Disease*, Chap. V, page 623, Edwards Brothers, Ann Arbor, Mich., 1937.

4 Howe, J. S.: "Daily Variations in the Tuberculin Recation, I. The Range of Variation in the Tuberculous and Control Subjects," *Amer. Rev. of Tuberc.*, 37: 264, 1938; II. "Relation to the Vascular Pressor Episode," *Amer. Rev. of Tuberc.*, 37: 273, 1938.

5 Petersen, W. F.; Howe, J. S., and Milliken, M. E.: "Weather and Resistance in Pulmonary Tuberculosis," *Amer. Rev. of Tuberc.*, 44: 377, 1941; Petersen, W. F.: "The Clinical Episode in Tuberculosis and the Meteorological Environment," *Amer. Rev. of Tuberc.*, 46: 407, 1942.

6 Kampmeier, R. H.: "Tuberculous Mediastinitis," *Amer. Rev. of Tuberc.*, 37: 71, 1938.

7 Petersen, W. F.: "Browning's Meteoropathological Observations in Tuberculosis," *Amer. Rev. of Tuberc.*, 46: 690, 1942.

8 Jones, K. P., and Tilden, I. L.: "Tuberculous Myocardial Aneurysm with Rupture and Sudden Death from Tamponade," *Hawaiian Med. Jour.*, 1: 295, 1942.

9 Pottenger, F. M.: "Living with the Weather," *Ann. Int. Med.*, 14: 502, 1940.

10 Mills, C. A.: "Body Combustion Rate as the Basic Determinant of Resistance to Infectious Diseases," *Urologic and Cutaneous Review*, 43: 817, 1939; "Value of Environmental Control in Tuberculosis," *Hospital*, 11: 51, 1937; "Susceptibility to Tuberculosis: Race or Energy Levels?" *Amer. Jour. Med. Science*, 189: 330, 1935.

Hazards of Pneumothorax*

DUANE CARR, M.D., F.C.C.P.**
Memphis, Tennessee

Pneumothorax in the treatment of pulmonary tuberculosis is unquestionably one of the great medical advances of all time. While its use has been popularized largely in the last three decades, we can hardly claim it as one of the achievements of our own precocious generation.

In 1819, Carson, a brilliant physiologist and physician, observed, "A hemorrhage from one of the lungs, a frequent prelude to consumption, if not immediately fatal, would certainly be stopped by the collapse of that lung." He points out that, "the diseased part would be placed in a quiescent state, receiving little or no disturbance from the movements of respiration which would be performed solely by the other lung, and the divided surfaces would be brought into close contact by the same resilient power which before had kept them asunder." He likewise saw the need of bringing the lung "to a state of collapse by degrees only."

Such is the fundamental conception of the function of collapse therapy, outlined 121 years ago, and little changed since. Much later, improvements in understanding of the physiological principles and the technic of pneumothorax progressed. A manometer was added to the equipment, the value and necessity of frequent fluoroscopic observations became appreciated, and a hazardous procedure was converted into a safe operation in the hands of competently trained physicians.

Today there is no need to draw up a brief for pneumothorax. Its universal use in every country in the world is proof of its value. In many major sanatoria as high as sixty-five per cent of the patients are receiving pneumothorax or have had it attempted. Out-patient clinics have been established for ambulatory patients to continue refills after being discharged from the sanatoria. Refills are administered in private offices in every city of size in the country, and in many towns and villages. It is the aim of the tuberculosis program in almost every state to have located in each community at least one physician qualified to give refills, so that sanatorium beds may be more quickly emptied to receive additional patients. Enthusiasm

---

*Read before the Twenty-seventh Annual Meeting of the Southern Tuberculosis Conference, Memphis, Tennessee, October 5-7, 1942.

**From the Department of Surgery, University of Tennessee College of Medicine.

for pneumothorax among the general medical profession and the laity is increasing until it seems that tuberculosis is no longer the dread disease it was once thought to be.

There is where the trouble starts.

The greatest hazard of pneumothorax is its apparent simplicity. It is true that the technic of inserting a needle between the ribs and into the pleural cavity of a patient with an already established pneumothorax is not difficult to master. Nor does it tax the ingenuity too much to learn the proper valves to turn on a pneumothorax machine to put a few hundred cubic centimeters of air into the pleural cavity. This fact has led many a practitioner, earnestly wishing to help his community, to buy a pneumothorax machine and proceed to give pneumothorax refills after visiting a clinic for a day or two, or even a couple of weeks, to see how it is done. If successful with the first few patients, he may even be encouraged to induce a few pneumothoraces for the indigent patients in his community who might have to wait for a sanatorium bed, or whose economic status would stand in the way of induction by an expert. If no immediately fatal accidents occur and he is giving of his time and substance to the care of the indigent, it seems only fair that he should branch out into pneumothorax work for the community as a whole, receiving compensation from those who can pay him.

It is not the purpose of this paper to quench the fire of enthusiasm for pneumothorax in the breast of any physician or layman interested in the control of tuberculosis. It is its purpose, however, to point out some of the pitfalls and hazards which lie in wait for the unwary or untrained manipulator of a pneumothorax machine, as well as some which are inherent in the method itself.

First of all, the administration of pneumothorax treatments is not synonymous with treating a tuberculous patient. To quote Barnwell, "The mere acquisition of technical skill in the performance of a pneumothorax is not an adequate preparation of the pneumothorax therapist. A broad knowledge of the natural history of tuberculosis under *various forms* of treatment, and also a familiarity with pneumothorax *complications and their treatment* are essential. Such training necessitates service in an institution actively employing pneumothorax. The indications for pneumothorax therapy are definite enough and the methods of observing and controlling it reliable enough to place it outside of the realm of 'clinical hunch'."

Perhaps the greatest danger in the use of pneumothorax is a desire to substitute it for bed rest and the other essential elements of a sanatorium regime. As an adjunct to be used in addition to rest, it is unexcelled in the group of patients to whom it is applicable; but it is no substitute and should not be used as such.

SELECTION OF CASES

It is generally agreed among phthisiologists that not all patients are suitable for pneumothorax treatment. Some do not need it. Others definitely should not have it, but require one of the other forms of collapse treatment. A number of patients who do need pneumothorax are found to have conditions existing, as the pneumothorax develops, which make the prompt abandonment of the treatment imperative. For example, the continuation of a pneumothorax which is only partially effective in closing a cavity leads only to disappointment, and in some instances to irreparable damage. It is far better to abandon it promptly and obtain a thoracoplasty or other appropriate form of collapse. Another example is the continuation of a pneumothorax induced on a patient with a large, thin-walled "tension cavity"—one which apparently becomes larger as the rest of the lung relaxes. The ball-valve mechanism in its bronchial outlet permits air to enter the cavity but neither air nor pus to escape. The pneumothorax frequently accentuates, rather than relieves, this tendency so that such cavities have been known to rupture into the pleural cavity and cause a virulent, and frequently fatal, mixed infection tuberculous empyema.

Prominent in the group of patients who should not have pneumothorax are those who have so-called "asthma," or those who wheeze. At least pneumothorax should be considered only when a bronchoscopic examination has been performed to rule out the presence of tuberculous tracheobronchitis. A pneumothorax collapse in a patient with this condition will produce an atelectasis of the lung with a collapse which can never be reexpanded, frequently with trapping of infection in cavities and subsequent severe sepsis. Under these conditions fluid usually forms in the pleural cavity, followed in a large percentage of patients by a tuberculous empyema.

ACCIDENTS

Accidents, both minor and major, can for the most part be avoided if the operator is aware of their possibility and pays heed to the principles by which they can be prevented.

Of minor importance, for example, is a patient's sensitivity to novocaine if, before beginning a course of treatment, the patient is tested for such an idiosyncrasy.

Alarming, but not serious, is the subcutaneous emphysema which results from the puncture of an adherent lung at the time of an initial induction attempt. The air which is coughed out into the tissues from such a puncture wound may cause subcutaneous emphysema from his scalp to his groin, but the air is gradually absorbed and no serious effects are to be anticipated. Experienced induction

technic will avoid creating a puncture wound of the lung of sufficient size to produce this distressing phenomenon.

It is disconcerting to the physician and difficult to explain to a patient when it is discovered that the pneumothorax which has been developing so nicely is all in the abdominal cavity. Granting that none of the abdominal organs have been punctured, and they will not be if an accepted induction technic is followed, no harm is done, for pneumoperitoneum is frequently used intentionally as a therapeutic measure. This accident can, of course, be avoided by the proper selection of the site of entry of the initial puncture.

Perhaps the most embarrassing accident, of minor significance if promptly recognized, is that of inducing a pneumothorax on the wrong side. Trusting to a physical examination alone, or to one's memory even after reviewing x-ray films, will certainly lead, sooner or later, to this mishap. Unfortunately, there are no external signposts to guide one. All trained collapse therapists, after possibly having made this mistake *once* in their lives, avoid such a hazard by having x-ray films before them while the pneumothorax is being induced.

Not all accidents accompanying pneumothorax induction can be so lightly passed over as those just mentioned. Perforation of the lung, with its attendant danger of air embolism, is an accident which every experienced man keeps constantly in mind when performing either an induction or refill. It is well known that air in a peripheral vein of an extremity such as an arm or leg will do no particular harm, since it passes through the right heart into the pulmonary artery and is filtered out of the circulation in the capillaries of the lungs. But even a minute amount of air in the pulmonary venous system is a very different story. Such air passes through the left heart and into the general circulation where it is free to block a coronary artery or one of the important arterioles of the midbrain, and therefore may be promptly fatal. This is unquestionably the mechanism of so-called pleural shock, and accounts for the majority of those occasional sudden deaths which occur while a patient is receiving a pneumothorax treatment. Fortunately, sufficient care on the part of the operator will avoid this complication also.

Even when air embolism does not occur following the puncture of a lung, tearing of the visceral pleura will often cause sudden and total collapse of the lung itself. This uncontrolled and rapidly developing collapse will cause severe distress to the patient and even serious physiologic embarrassment which must be promptly recognized and immediately relieved.

Needless to say, one of the worst accidents which may befall a patient is to have infection introduced into the pleural cavity by

the pneumothorax operator. A pyogenic empyema makes drainage mandatory, with reexpansion of the lung, obliteration of the pleural cavity, and complete loss of the collapse. Such a patient who still needs relaxation of his lung must then resort to a thoracoplasty or other major surgical procedure. Fear of this accident causes the pneumotherapist to use every precaution to insure absolute sterility in this technic, including sterile tables, instruments, drapes, and gloves. Less rigid technic, even when no actual empyema develops, tends to cause patients to have "reactions" following pneumothorax refills, which are quite unnecessary.

COMPLICATIONS

Complications of pneumothorax cannot always be avoided, but to the informed collapse therapist they can be quickly recognized and, for the most part, readily overcome if attacked promptly.

Spontaneous collapse can occur due to the rupture of an adhesion, especially during the first few days of induction, or tearing of the lung due to the traction of adhesions. As in the case of the spontaneous pneumothorax due to puncture of the lung, prompt recognition and correction by the withdrawal of air will relieve the patient of his embarrassment, but delay may cause the patient to go into a state of physiological shock which no amount of stimulant, oxygen, and shock treatment will correct. It is even more confusing to the neophyte when a purely spontaneous pneumothorax of the *opposite* lung occurs during the course of pneumothorax treatment. Its significance is tremendous considering the fact that the loss of the use of the good lung is superimposed upon the partial loss of that of the intentionally collapsed lung. As these complications occur chiefly during the period of induction and development of a pneumothorax, it is important that the early portion of pneumothorax treatment be carried out in a hospital or sanatorium.

The loss of a pneumothorax space due to either insufficient or too infrequent treatments should hardly be considered a complication of pneumothorax. It will not occur at the hands of an experienced operator who uses his fluoroscope regularly and whose patients are cooperative and reliable in their attendance at his clinic. The tragedy, when it does occur, is that once the lung has contacted the chest wall and adhered, the space can more than likely never be recovered.

Another complication which will not occur under conventional management is that of mediastinal hernia, a condition in which part of the lung on the affected side is pushed across the midline in front of or behind the heart into the opposite hemithorax. Such a hernia can readily be recognized in a good fluoroscopic examination and promptly be corrected if present. However, it has been known to

escape attention until the mediastinal pleura ruptured following an excessively large refill, resulting in the death of the patient.

Probably the most consistently encountered complication of pneumothorax is pleural effusion, varying all the way from a few cubic centimeters of transient fluid which spontaneously disappears and has little or no significance, to those massive effusions which are persistent and require intensive and faithful treatment. No operator is entirely free of this complication, although the frequency with which it occurs varies widely in different hands. Factors which have been suggested as causative agents are: (1) too rapid induction, (2) positive pressures, (3) tension on adhesions, (4) lesions in or just beneath the pleura, (5) introduction of organisms insufficient to cause an empyema but enough to cause pleural irritation, and (6) introduction of novocaine or other foreign chemical into the pleural cavity. To assert my opinion regarding the relative importance of these or other possible factors is not the function of this paper. Suffice it to say that effusions occur, can usually be overcome if treated intensively enough, and are an indication for abandonment of the pneumothorax with substitution of other forms of collapse if they are persistent after adequate attempts to eradicate them.

In many instances persistent fluid will automatically cause the loss of a pneumothorax space by an adhesive pleuritis, a progressive creeping of the lung up the chest wall until no pneumothorax exists. This is unfortunate if the pneumothorax has been present too short a time to permit healing of the lung. Other forms of collapse must be substituted.

Another unhappy effect of persistent fluid is its tendency to become infected with the tubercle bacillus causing a tuberculous empyema. This is another complication which must be recognized and treated promptly. While many tuberculous empyemas may be cured, one which is persistent becomes a very real danger to the patient's health and life, and should cause the pneumotherapist to abandon his pneumothorax for another form of collapse therapy.

The final complication which will be mentioned is the lung which will not reexpand. Several conditions may contribute to this situation. A bronchopleural fistula, caused originally by the puncture or rupture of the lung, will prevent a lung from reexpanding when the desirable time comes. Tracheobronchial tuberculosis with stenosis of the bronchus will do the same thing. Or if pleural fluid has been permitted to remain in the pleural cavity for months or years, the pleura itself will become thick and tough like the jacket of a football and limit the lung to its reduced size forever. Some permanently contracted lungs can be avoided if the physician is able to foresee the factors which lead to their development. In any event, when

this complication does arise, the addition of some type of surgical plastic operation is indicated promptly.

CONCLUSION

In summary, it is the purpose of this paper to point out that the management of a patient with tuberculosis consists of many considerations besides that of technical ability to give a pneumothorax refill. Pneumothorax is an important, valuable and safe adjunct to the treatment of tuberculosis when used in properly selected cases, induced by an experienced hand, and when the patients are reviewed periodically by one who can recognize the various complications which may arise and institute appropriate treatment promptly.

Ideally, the induction of pneumothorax is a sanatorium or hospital procedure. To be commended are the efforts of general practitioners to obtain training in the management of tuberculosis, a need which is far from satisfied. For those who receive anything less than a year of service in a tuberculosis institution actively employing pneumothorax, it is strongly recommended that their efforts be confined to giving refills only, and that they accept the help and advice of a qualified sanatorium staff, or expert, in the induction of the pneumothorax, and, by periodic check-up examinations, the management of each case.

CONCLUSIONES

En resumen, el propósito de este trabajo es el indicar que el tratamiento del paciente tuberculoso incluye muchas otras cuestiones además de la abilidad técnica para administrar un relleno neumotorácico. El neumotórax es un coadyuvante importante, valioso y seguro en el tratamiento de la tuberculosis cuando se emplea en casos debidamente seleccionados, cuando lo inician manos expertas y cuando analiza periódicamente a los pacientes alguien con habilidad para reconocer las varias complicaciones que pueden sobrevenir a fin de aplicar prontamente el tratamiento apropiado.

Lo ideal es que la iniciación del neumotórax se lleve a cabo en un sanatorio o en un hospital. Son dignos de encomio los esfuerzos que hacen los médicos de práctica general para obtener adiestramiento en el tratamiento de la tuberculosis, una necesidad que todavía deja mucho que desear. Pero a aquellos que hayan recibido menos de un año de aprendizaje en una institución dedicada a la tuberculosis y que emplee el neumotórax energéticamente, se les recomienda vehementemente que limiten sus esfuerzos a dar rellenos solamente, y que acepten la ayuda y el dictamen del competente cuerpo médico de un sanatorio, o de un especialista, en la iniciación del neumotórax y, por medio de reexámenes periódicos, en el tratamiento de cada caso.

899 Madison Avenue.

Thoracoplasty With Apicolysis*

E. W. COYLE, M.D., F.A.C.S., F.C.C.P.**
San Antonio, Texas

I. *Historical*—For the last three centuries surgeons have attempted to check the course of cavernous pulmonary tuberculosis. This attempt has been carried out by different procedures, including opening of the cavitation so as to provide drainage to the exterior or in order to have an available route for the application of medications; also removal of the diseased portion of lung has been attempted and in more recent years success has been obtained in various measures designed to exclude the involved organ from active function by the introduction of air into the pleural spaces *or by the partial excision of the thoracic cage.* It is this last achievement that I will try to outline in this paper from our present knowledge of pathology, physiology, and surgical technique.

In 1888 Quincke and Spengler made the original proposal of rib resection later carried out though unsuccessfully by Bier and Mikulicz. It was Friedrich in the 19th century, in association with Bauer, who first reported a successful thoracoplasty consisting of the removal of long segments of the *2nd to 9th* ribs with periosteum and intercostal muscles. The operation produced a marked degree of shock and carried a high mortality although those who survived it obtained satisfactory results. Later Wilms and Sauerbruch developed the operation of paravertebral thoracoplasty. independent of each other. They removed shorter posterior segments of the ribs at first and later resected up to five inches or more of the lower ribs and shorter segments of the upper ribs.

The modern extrapleural thoracoplasty is therefore based upon the Wilms-Sauerbruch technique.

II. *Indications and Contraindications*—Indications for thoracoplasty resemble in a general way those for the introduction of gas into the pleural space, with certain *important* additions. The candidate for thoracoplasty must first, last, and always be in such physical condition as to permit a major surgical intervention without risk of immediate collapse. Of special importance is the condition of the myocardium and the circulatory system as an entity. The respiratory system also must be adequate, as the healthy breathing space must compensate for the diseased tissue, particularly during the surgical procedure. The lesion under treatment

---

*Presented at the Annual Meeting, Texas Chapter, American College of Chest Physicians, Houston, Texas, May 11, 1942.
**From Woodmen of the World Memorial Hospital.

should preferably be a unilateral lesion or, at least, the contralateral lesion must be under control. Whatever the lesion might be clinically, it must be stabilized; therefore, an exudative lesion, if present, should of course be apical or at least in upper lobes. All other available means of collapse therapy should first be proved unsuccessful.

Post mortem studies in large series of cases have revealed the impossibility of obtaining a satisfactory one hundred per cent collapse of a tuberculous cavity; therefore, it is thought that it is not the collapse of a cavitation which brings on satisfactory clinical results, but rather a kinking of the bronchus leading to the area in which the cavitation is present. This kinking of the bronchus renders that portion of lung tissue atelectatic and further adds to the closure of the cavity. For this reason cavities in the apex and near the periphery of the lung, as a rule, are greatly benefited by thoracoplasty.

It is bad judgment to recommend this procedure merely because nothing else is possible and because there is a feeling that since the patient may die without the operation, he may as well be given the chance of a favorable result. This was true in three cases in our series. This type of patient will almost always die as a result of the operation and the fatality thus detracts from any statistical study in properly selected cases. Furthermore, patients over fifty years of age are, as a rule, poor risks because their myocardial reserve, which is very difficult to estimate pre-operatively, is usually low. Certain extrapulmonary lesions are definite contraindications for the performance of a thoracoplasty.

III. *Preoperative Preparation and Study*—Patients who are considered candidates for a thoracoplasty are studied over a period of weeks in order that one may obtain an idea of the general condition of the patient. The temperature, pulse, and rate of respiration are seriously considered; the amount of expectoration and relative concentration of acid fast organisms are likewise taken into consideration. The presence of an anemia is ruled out, as well as the possibility of any pathologic involvement of the circulatory system. Electrocardiographic tracings are obtained, as well as a history of past decompensation; the vital capacity is taken at periodic intervals in order that we may estimate the breathing surface of the lungs. The candidate and close relatives are then told of the advisability of thoracoplasty and definite plans are made to carry out the procedure. We attempt to have at least two compatible donors available at the time of operation and we strongly recommend that this procedure not be attempted unless at least one compatible donor is present at the hospital during the operation. The patient is told to expectorate all sputum possible the morning before surgery. A sedative is given at bedtime and repeated the next morning.

Morphine and atropine are administered about 45 minutes before the operation and the patient is taken to the operating room shortly after the noon hour. Oxygen is available in sufficient quantity for postoperative use.

IV. *Anesthesia*—In our hands intratracheal cyclopropane has been the anesthesia of choice as it provides the patient with an open airway through which respiration is controlled at will. A slow, careful induction usually insures a smooth course during the operation. As a rule, sufficient relaxation is obtained from cyclopropane alone, though occasionally when the pleura is separated from the chest wall during the apicolysis, such stimulation results, that irregular and often paradoxical respiration ensues, rendering the procedure more difficult and perhaps harmful to the patient. Administration of ether with a closed system is likewise satisfactory.

V. *The Operation*—In our group of one hundred operations covering a period of three years, we have followed the practice of removing the entire 1st, 2nd, and 3rd ribs with occasional removal of part of the fourth rib at the 1st stage operation. In addition we do an apicolysis dropping the apex of the lung to the level of the 4th or 5th rib posteriorly. The intercostal muscles, vessels, and nerves are then severed posteriorly and ligated. The transverse processes and rib stumps are not removed in all cases, as we believe that this helps prevent subsequent scoliosis. We then attempt to fasten the apex by suturing the intercostal muscle bundles and periosteum to the first intact rib, thus forming a cap over the apex which holds it firmly fixed and inhibits the apex from rising during the first three to four weeks following operation.

The wound is then closed without drainage and the large amount of dead space thus created fills with serum and aids in keeping the apex depressed and provides a collapse of the cavities from above downward and frequently displaces the cavities downward and somewhat anteriorly away from the costovertebral gutter.

Usually, in the case of large cavities or multiple cavities, a second stage is necessary. However, there is definitely a place for the one-stage thoracoplasty with apicolysis. Those cases presenting high apical cavities with fibrotic retraction of the trachea to the affected side quite frequently obtain collapse of the cavitation and in a short time the sputum is rendered negative, with the one stage procedure.

In the second stage operation the entire segment of the first remaining rib is removed and then shorter segments of the next two or three ribs are excised, giving us a horizontal collapse of the upper half or two-thirds of the chest cavity and yet leaving adequate space for the functioning lower lobe. During the latter part of the operation a whole blood transfusion is given and the patient is then returned to his room.

VI. *Postoperative Care*—The most immediate and pressing necessity is the prevention of shock. This we attempt to do by restoration of blood volume and the use of cardiac stimulants if necessary (50% glucose, coramine, caffein, sod. benzoate, etc.). It is important to watch the patient closely for serious postoperative hemorrhage. Though rare, cases of death from this cause in what, at the operation time, was considered "a good technical job," have been reported. The dressings applied are light although compression of the infraclavicular space is necessary in order to prevent paradoxical respiration which, as will be mentioned, is of a harmful nature. No opiates are administered so as to avoid suppression of the cough reflex, although on few occasions we have resorted to small doses of dilaudid (gr. 1/64 every 6 to 8 hrs.) in order to relieve pain and obtain rest. Intranasal oxygen is given when indicated by irregular or fast and shallow respiration or when any degree of cyanosis is evident. Expectoration is encouraged, as well as deep breathing exercises in the sitting position, in order to prevent the occurrence of atelectasis. Sedatives and intravenous fluids are given according to the needs of the individual case. The diet is restored as soon as the patient's general condition warrants its administration.

VII. *Complications*—Thoracoplasty must be considered a potential shock-provoking operation; therefore, this complication should be kept in mind and necessary steps should be taken to prevent it. The watchful and diligent study of the vital signs (temperature, pulse, blood pressure, respiration) together with the appearance of the patient are the means by which shock is detected. Shock *per se* may be due largely to one of two causes: (1) The extensive surgical excision of part of the thoracic cage with its imperative accompanying trauma, and (2) hemorrhage during or immediately following the operation. Oftentimes both factors play a vital part in the causation of shock. Radiographic studies of the chest are of value in the determination of intrathoracic hemorrhage which may be into the extrapleural space or into the intrapleural or visceral space. Circulatory collapse of myocardial origin should be differentiated from the above condition and from a mediastinal flutter.

Atelectasis is the second most important complication of thoracoplasty and most authors state that it occurs with greater frequency in those cases in which the apicolysis is performed. The diagnosis of this condition is sometimes difficult and, therefore, is often missed. The onset may be confusing, with prolonged shock and respiratory distress, but the physical signs of dullness or flatness on percussion, limited thoracic excursion, absent or distant breath sounds and, finally, the characteristic radiographic appearance, complete the diagnosis. As to the etiologic factor for this condition, it may be endobronchial or exobronchial. When the former factor

plays a part, bronchoscopy (followed by aspiration) is a life saving procedure provided the instrument may be passed as low as the obstruction is present.

An aspiration pneumonia should always be kept in mind and its prevention is of great benefit; for this, expectoration is encouraged as well as deep breathing exercises and in wet cases the anaesthesist attempts to remove all secretions by aspiration through the intratracheal tube before the patient is removed from the operating table.

Extension of the tuberculous lesion often occurs in cases not selected properly. Often the increase is bronchogenic and as a rule involves the same side though at times a contralateral spread occurs.

Infection of the apical space or of the wound occurs occasionally. This may be prevented to some extent by the removal of all dead or devitalized tissue, by adequate hemostasis, by the conservative use of suture material and by irrigation of the wound before the closure. We do not advocate the use of sulfanilamide powder in a clean wound of this nature. The presence of an unexplainable draining sinus over a long period of time with questionable or atypical radiographic plates should lead one to think of the possibility of a foreign body being left within the space.

Paradoxical respiration should be prevented as it contributes to hemorrhage and spread of the infection, and increases the myocardial strain without advantage. This is prevented by the capping of the lung as described in our technique and by the application of a firm padding over the infraclavicular region, held in place by tight adhesive strapping.

There are no doubt countless other complications; however, the most important have been given consideration in this paper.

STATISTICAL REVIEW

During the last three years we have performed a total of one hundred operations with ten deaths, giving a gross death rate of 10 per cent. Of these one hundred operations, sixty-one were first stage with excision of the 1st, 2nd, 3rd and in some cases part of the 4th rib. In this group we lost six, giving a death rate of 9.8 per cent. Of these sixty-one cases, thirty-eight warranted a second stage operation due to more extensive involvement of lung tissue necessitating further collapse. In this second group we lost four cases with a death rate of 10.5 per cent. One case needed a third stage for the excision of the 8th, 9th and 10th ribs due to advanced involvement of the lung complicated by a tuberculous empyema.

In our series we have had the unfortunate experience of complications such as gross hemorrhage into the intrapleural space with death on the eighth postoperative day. This particular patient, though a good surgical risk and with a relative atraumatic surgical

procedure and good hemostasis at time of closure, had a slow though persistent hemorrhage into the intrapleural space which without doubt greatly contributed to his death on the eight postoperative day (type I blood; unable to get compatible donor).

One case done on a 56-year-old white male resulted in a postoperative right heart failure though the surgical procedure was short and immediately successful. This patient died on the fourth day with a typical picture of right heart failure even though preoperative studies revealed a satisfactory condition. We, therefore, would like to emphasize strongly that cardiac reserve is very difficult to estimate, particularly in an elderly individual who has been bedridden for some time, although his physical condition appears to be good.

Unfortunately, we are unable to state the therapeutic end results in our series of cases in regard to improvement of the tuberculous condition. This we are unable to do because the majority of our patients are not followed for a number of months after operation. The cases are returned to the various institutions where they were originally studied—the Local County Tuberculosis Clinic, the Woodmen of the World Hospital—and to the various parts of the country from which they were referred.

We would like to emphasize once again the importance of a correct recommendation for this type of surgery, as we now recognize the fact that three of our death cases were hopeless from the beginning and only had their operation as their last and only chance for improvement.

CONCLUSIONS

1) Surgical treatment of pulmonary tuberculosis, though by modern means a relatively safe surgical procedure, has been present in the annals of medical literature for three centuries.

2) The indications and particularly the contraindications to this operation have been reviewed.

3) The proper preoperative status of the patient, as well as the correct recommendation for thoracoplasty, are of great importance.

4) Our technique has been outlined and though we claim no originality in any part or portion thereof, we believe this to be the procedure of choice in properly selected cases.

5) Complications should be prevented rather than treated, and careful postoperative study of the patient will, in the majority of cases, reveal the possible diagnosis.

6) A series of one hundred thoracoplasty operations are hereby reported with a gross death rate of 10 per cent. These operations have been analyzed as to stages and respective death rate.

CONCLUSIONES

1) El tratamiento quirúrgico de la tuberculosis pulmonar, que llevado a cabo por métodos modernos es un procedimiento quirúrgico comparativamente seguro, ha aparecido en los anales de la literatura médica desde hace ya tres siglos.

2) Se ha repasado las indicaciones y particularmente las contraindicaciones de esta operación.

3) Tanto el exacto estado preoperatorio del paciente como la correcta recomendación de la toracoplastia tienen gran importancia.

4) Hemos delineado nuestra técnica, y aunque no pretendemos que ninguna parte o porción de ella ha originado con nosotros, sí creemos que éste es el procedimiento de elección en casos correctamente seleccionados.

5) Es preferible prevenir que tratar las complicaciones y, en la mayoría de los casos, la cuidadosa observación postoperatoria del paciente descubrirá el posible diagnóstico.

6) Preséntase aquí una serie de cien toracoplastias con una mortalidad total de 10 por ciento. Se han analizado estas operaciones en sus diferentes etapas con sus respectivas mortalidades.

1005 Nix Professional Bldg.

Tuberculosis of the Spine Complicating Pulmonary Tuberculosis

HUGH M. KINGHORN, M.D., F.C.C.P.
Saranac Lake, New York

What will be said in this communication will not be confined to the spine, but will also include the pelvis and hips, because any study of a diseased condition in any of these three areas should embrace a thorough study of all of them.

Tuberculosis of the spine is quite frequently not localized to one vertebra. Sometimes several vertebrae separated at some distance from one another are also involved. If, therefore, the site of the symptoms be the only one studied, other areas of disease may be overlooked. This rather frequently happens.

We wish also to emphasize not only the importance of a thorough study by the usual clinical methods, but particularly by x-ray examination of all three areas. The x-ray examination should always include front and lateral views of the entire spine, pelvis and hips. Where possible these views should be made in stereo. The cases that will be recorded deal with this form of tuberculosis in the adult and not with children. All of the patients had also advanced tuberculosis of the lungs. Most of them seemed hopeless, both as regards the bone disease as well as with the lung condition. Operative interference could not be considered on account of the extensive lung involvement. Mechanical rest treatment was therefore employed in all of them. This rest treatment was complete, and gave evidence of the astounding results which can be obtained by it with associated bone and lung tuberculosis. This is not surprising, if one thoroughly understands the mechanism of the treatment of tuberculosis by rest. We believe, however, that probably the best treatment for these spinal conditions in patients whose lungs are clear is operative fusion.

It is an unfortunate fact that many physicians have not grasped the greatest single factor in the treatment of tuberculosis, namely, the proper employment of rest. Immobilization of the diseased area is an extremely important curative measure in both surgical and medical tuberculosis, and pathology gives us conclusive support to the treatment which endeavors to obtain functional rest to the tuberculous area.

The most favorable conditions for healing tuberculosis occur when blood and lymph become slighter in the diseased areas, when

Received for publication November 9, 1942.

the washing out of bacteria and bacterial products is restricted, and thus the danger of metastatic formation in healthy areas is lessened. If blood and lymph circulation in a tuberculous area reach a minimum, the formation of toxin diminishes enormously. The healing action of rest is thus explained.

The earlier the detection of tuberculosis of the spine and pelvis and hips, or of tuberculosis anywhere in the body is made, the more successful will be its treatment by appropriate rest. If disease of these areas were recognized sooner than it usually is, most of the cases would be cured by properly applied rest treatment. If the disease be allowed to proceed until deformity is seen, as in spine and hip-joint disease, the difficulties of the patient are greatly increased, and the prospect of a favorable result much diminished, though not destroyed.

With an inflamed joint we can usually detect its condition by the presence of a single local symptom, namely, increase of temperature in the parts. In disease of the spine there is one symptom which almost always stands out most prominently, namely, local and fixed pain upon the surface of the body, with or without exacerbations, and absence of local increase of temperature at this spot. By means of this symptom, we may recognize a morbid condition of the spine long before there is any evidence of disease by deformity.

Tuberculosis of the spine may begin in the vetebrae or in the intervertebral substance, probably most frequently in the intervertebral substance, or where this is joined to the vertebra. This disease of the spine is often the result of accident, because with accidents the most frequent lesion is a partial severance of the vertebra from the intervertebral substance. The pain associated with diseased spine is found upon the skin supplied by the nerves which escape from the vertebral canal through the intervertebral foramina close to the bones or intervertebral substances, either of which may be the site of the disease. The recognition and correct interpretation of the cause of this pain upon the surface of the body should give us the best opportunity for early and correct diagnosis in tuberculosis of the spine. When a patient complains of pain on the surface of the body, it must be produced by the nerve which is located there, and somewhere in the course of its distribution between its peripheral termination and its central origin (either spinal or cerebral), the precise cause of the pain located on the surface must be situated.

John Hilton[1] stated the following (p. 91): "Superficial pains on both sides of the body, which are symmetrical, imply an origin or cause the seat of which is central or bilateral; and unilateral pain implies a seat of origin which is one-sided and as a rule exists on the same side of the body as the pain."

With children almost all diseases of the spine are the result of slight accidents overlooked. No cases of diseased spine are so immediately dangerous to life as those in the upper part of the cervical region, especially if situated between the first and second cervical vertebrae.

Hilton also stated the following (p. 168): "The same trunks of nerves, whose branches supply the groups of muscles moving a joint, furnish also a distribution of nerve to the skin over the insertions of the same muscles; and—the interior of the joint receives its nerves from the same source."

Let me illustrate: The anterior crural nerve supplies nearly all the muscles which we use in the first effort of progression; we flex the thigh, we extend the knee, and we slightly evert the leg. Nearly all the muscles used for these purposes (the psoas, the iliacus, pectineus, crureus, subcrureus, the two vasti, the rectus, the sartorius muscles) are supplied by the anterior crural nerve. So also is the skin over their insertion, as well as the joints which they move, supplied by this same nerve. The numerous cutaneous nerves are branches of the anterior crural, and supply the skin as far as the knee joint, and are distributed over the thigh, and include the external cutaneous nerve, and correspond with the subjacent distribution of the anterior crural nerve to the various muscles. Of these various cutaneous nerves I mention the long saphenous which runs down the leg as far as the ankle joint and the side of the foot. If we dissect the inner side of the knee and leg, we see that the sartorius muscle is intimately and largely attached to the fascia of the leg. In fact the fascia of this part of the leg must be considered as part of the insertion of the sartorius. Not only is the sartorius supplied by the anterior crural nerve, but the fascial insertion, extending down the leg even as far as the ankle-joint, where it is blended with the fascia over the foot on the inner side of the internal malleolus, receives its nerve supply from the same source. This explains why the long saphenous nerve, which is derived from the anterior crural, should pass through the fascia of the thigh, become subcutaneous, and then follow the course of the skin as far as the inner side of the ankle-joint, sometimes as far as the inner side of the dorsal aspect of the foot, thus following the fascia. Thus when one examines this fascial association, one sees that it is only carrying out the principle that the same nerve which supplies the muscle supplies the skin over the insertion of the muscle, the sartorius having a long fascial insertion as far as indicated, and requiring an equally long nervous supply.

The anterior crural nerve sends branches also to the hip-joint. Filaments of this nerve can be traced to the anterior part of the capsular ligament of the hip-joint, and it sends branches to the

muscles of the thigh, and to the capsular ligament of the knee-joint.

This nerve thus supplies the joints which its muscles move, the anterior part of the hip-joint and a portion of the knee-joint. It also supplies the skin over the whole of the muscles of the thigh, as well as the skin on the inner side of the leg, as low down as the limits of the fascia to which the sartorius muscle is attached.

In tracing the nerves it is important to give to them some useful reference. Thus the long saphenous nerve follows the course of the saphena major vein, and this is probably why patients feel so much pain when these veins are dilated and press upon portions of the nerve.

So with the course and distribution of the obturator nerve. The trunk of this nerve comes out of the vertebral canal between the third and fourth lumbar vertebrae. It arises from the third and fourth lumbar nerves, and usually receives an additional root from the second. The spinal marrow ceases opposite the second lumbar vertebra. There is no marrow below the second lumbar vertebra in the adult. The nerve then passes down through the pelvis, and it may send some filaments to the sacro-iliac articulation. It lies close to this articulation and possibly suffers from its nearness to it when diseased. It then passes across the brim of the pelvis and along its inner aspect, then over the obturator internus muscle, through the obturator foramen, where it gives a branch to the obturator externus muscle. The obturator nerve then gives a filament to the capsular ligament, branches downward to the notch of the acetabulum, and one filament goes to the hip-joint to be inserted into the base of the ligamentum teres. This explains the sympathetic pains associated with the hip-joint which occur at the knee-joint, because the obturator nerve also supplies the knee-joint. Having sent branches to the capsular ligament and to the ligamentum teres we now see the muscular distribution of the nerve. It supplies the obturator externus and the adductor brevis. Filaments pass to the adductor longus, and to the adductor magnus, and to the gracilis. One sensitive filament passes down to the inner side of the knee-joint, and the other (a part of the posterior division) goes through the adductor magnus into the popliteal region, and there enters the knee-joint at its posterior part. The distribution of this nerve is another illustration of the same nerve supplying muscles and also joints moved by them.

I have gone into the origin, course and distribution of this nerve for a special reason. Due to the wide distribution of this obturator nerve, it is apparent that there are many different causes for pain felt along its branches to the inner side and interior of the knee-joint. I wish to again mention the great value of accurate obser-

vation as regards the locality of pain on the surface of the skin. If we trace the trunk of the obturator nerve, we shall find it associated with many internal and external parts. If we assume the obturator nerve to be the seat of pain on the inner side of the knee, and within the knee-joint, we see that this may depend upon disease within the vertebral canal, or it may depend upon some diseased condition of a vertebra near which the nerve lies. This nerve passes over the sacro-iliac joint, and when that joint is inflamed, patients may complain of pain within the knee-joint and on its inner side. A patient may complain of pain on the inner side of the knee when the disease is not in the hip-joint, but in the sacro-iliac joint, although the symptoms may point to hip-joint disease. As the obturator nerve proceeds downwards, a psoas abscess may produce the pain in the knee. On the left side of the body this nerve runs under the sigmoid flexure of the colon, and the colon, when distended by faeces or diseased by cancer, may produce pain on the inner side of the knee.

We know that hip-joint disease can produce pain on the inner side of the knee. This disease begins frequently in the ligamentum teres. Since one branch of the obturator nerve goes to the hip-joint, a second to the interior of the knee, and a third to the inner side of the knee, we see how disease in the interior of the hip-joint can produce reflex pain on the inner side of the knee, and in the interior of the knee-joint.

It is this knowledge of the course and distribution of these nerves that has helped me on different occasions to suspect and finally to diagnose disease in the lower spine and in the sacro-iliac region and hips.

The same method should be applied in examining the cause of pain on the inner side of the knee. Three nerves from three different sources appear here—the obturator, the anterior crural, and the great sciatic. If a patient complains of pain on the inner side of the knee-joint, we should try to find which nerve produces the pain. There are three lines of direction for this pain, and we should examine these three lines of direction to ascertain in the course of which nerve the cause is situated. Thus we should examine the anatomical relations of the obturator, the anterior crural and the great sciatic to find out, if possible, the real cause of pain felt on the inner side of the knee-joint, because a disturbing cause associated with the course of any of these nerves might produce the pain.

From what has been said, it is seen that there are a number of nerves located around and within the knee-joint, and that these nerves are derived from many sources. The great sciatic sends branches not only through the popliteal space to the posterior part

of the knee-joint, but its external popliteal branch supplies deep filaments to the outer side of the knee-joint, both above and below the inter-articular space.

OBJECTIONS TO PROLONGED REST TREATMENT

We quite frequently meet with objections when we advise prolonged rest treatment for lung or spine tuberculosis. For either of these conditions prolonged rest treatment is necessary, if we wish to obtain a permanent arrest of the disease. A principal objection is that the prolonged rest in the completely recumbent position will produce a lessening of the general health, and that the muscles will become soft and flabby. To this objection I can say positively that I have never yet seen a case of advanced lung or spine tuberculosis or a combination of both in which the health has deteriorated as a result of rest treatment. On the other hand, these cases have invariably greatly improved in their general health.

Another objection is that the joints, their soft parts being kept in restraint and not being used, although not at the time showing actual disease, may undergo serious structural change and deterioration by prolonged rest, and that even healthy joints may become ankylosed. This is not correct. There may be a temporary alteration and a diminished elasticity of the articular cartilage. The ligaments also may become feeble. Bones may not be so firm and muscles may lose their strength from disuse. Such effects, however, are temporary. Reparation in all these structures is brought about by careful re-employment, and later these structures show no defects.

HIP-JOINT DISEASE

It is important to diagnose hip-joint disease early. This is also called morbus coxae, or morbus coxarius. It is the inclination of surgeons to think that the patient with hip-joint disease should have certain marked symptoms which are considered as characteristic, such as shortening or lengthening of the leg, a fullness or flatness over the gluteal region, lack of symmetry in the sub-gluteal folds, pain at the inner side of the knee, disurbed health and a good deal of lameness. These symptoms, however, may fail as positive indications. For instance, lengthening and shortening of the leg in the early stage of the disease is nearly always the result of inclination of the pelvis. This does not refer to cases with advanced disease about the joint, nor to cases of dislocation. These characteristic symptoms which are given as pointing to disease of the hip-joint are not early symptoms. When such symptoms occur, the disease in the joint is considerably advanced. It is therefore important that the diseased condition should be recognized previous to

that period, because that is the time when the best results will occur from long continued rest treatment.

It is a serious mistake for us to assume that there is nothing wrong in the hip-joint, unless some of the above-mentioned prominent symptoms be present. The most important point is the early detection of the first deviation from a healthy state. Even a suspected state of disease justifies rest treatment. It is usually possible to elicit slight degrees of spasm, even in the early stages.

The muscles of the hip-joint act in groups, and each group has a nerve trunk of its own, and each nerve gives off a branch to the hip-joint. For instance a branch of the anterior crural nerve goes to the hip-joint; a branch of the obturator nerve goes to the capsular ligament and to the ligamentum teres, and a branch goes to the posterior part of the hip-joint from the sacral plexus which supplies the gemelli, the quadratus femoris and the obturator internus. This knowledge of anatomy explains how the remote and sympathetic pains associated with inflammation or chronic disease of the hip-joint, are not always found at the same area of the leg. In some cases of hip-joint disease, one of the earliest symptoms is distant from the actual seat of disease, namely, pain within the knee, or on the inner side of the knee-joint. The explanation of this is that the obturator nerve, which gives off a branch to the ligamentum teres, sends a branch to the interior of the knee-joint, to the inner side of the knee-joint, and sometimes lower down. The inflammation or disease of the ligamentum teres embraces the small branch of the obturator nerve, and sympathetic pain is felt at the other end of this nerve, on the inner side of the knee or within the knee-joint.

Though this frequently occurs with the obturator nerve, it should sometimes occur with other nerves of the hip-joint. The frequency of this knee, pain, whether on the inner side of the knee or within the knee-joint, indicates that the ligamentum teres is the most common seat of early disease. This knowledge of knee pain has repeatedly helped me to suspect early tuberculosis either in the hip-joint, sacro-iliac joint or lower lumbar region.

This sympathetic or reflected pain is also observed in old people who have had a doubtful slight injury to the hip. Sometimes the ligamentum teres softens and disappears. These persons may complain of severe pain within the knee-joint or on its inner side.

If the anterior part of the capsular ligament (which receives a branch from the anterior crural nerve) is inflamed, a patient with disease in the hip-joint may have pain on the front of the knee, or on the inner side of the ankle, because branches from the anterior crural go to these areas. And if the condition occurs at the posterior part of the capsular ligament, which gets branches from the sacral plexus, the patient may have reflex pain at the heel, or in the foot.

It is sometimes thought that the reflex pain of disease in the hip-joint is always on the inner side of the knee-joint, and that this local symptom is necessary for a correct diagnosis. This is probably not correct. It does more frequently occur, because the ligamentum teres is likely the location where hip-joint disease most commonly begins, and this corresponds with the frequent finding of reflex pain on the inner side of or within the knee. It occasionally happens in hip-joint disease that there is no pain in the knee-joint. This local pain, therefore, within or at the inner side of the knee-joint is not a constant symptom, and not always to be relied upon as indicating disease of the hip-joint.

Particularly with disease about the hip-joint or sacro-iliac region, it is necessary to take great care to make accurate measurements of both legs. The patient should lie flat on his back on a firm mattress, with the legs parallel and extended. One should then feel for the point of the anterior superior spine of each os innominatum, and mark the overlying skin very accurately. Measurements should then be taken from the above mentioned points of bone, along the inner side of the patella, down to the transverse ridge which can be felt upon the inner surface of the internal malleolus and just above its tip.

TUBERCULOUS SACRO-ILIAC DISEASE

Disease of the sacro-iliac joint or of the lumbar vertebrae is sometimes mistaken for hip-disease. In all three conditions reflex pains are usually felt down the leg to the knee, and even to the foot. X-ray examination is of the greatest help to locate the exact site of trouble. Powerful ligaments hold the sacrum to the os innominatum on each side, and strong ligamentous tissues occur between the lateral aspect of the sacrum and the inner and posterior part of the os innominatum.

The sacro-iliac articulation or synchondrosis, like the symphysis pubis, is an amphi-arthrodial joint, but differs from it, inasmuch as it has an interosseous ligament, as well as an interosseous or symphysial cartilage. By amphi-arthrodial is meant a form of articulation in which the bones are connected by intervening substance admitting slight motion; symphysis. The bones which enter into the sacro-iliac joint are the sacrum and ilium. The articular surface of each bone is covered with a thin plate of cartilage, thicker on the sacrum than on the ilium. These cartilaginous plates are in close contact and are united together by irregular patches of softer fibro cartilage, and at their upper and posterior part by fine interosseous fibers. In a considerable portion of their extent, especially in adult life, they are separated by a space containing a synovial-like fluid. The joint is very large and very firm, and held together by liga-

ments and not muscles, and possesses only slight motion. At every step it bears the whole body weight, and transmits it through the pelvis to the leg.

If disease should occur at the sacro-iliac joint, the patient will probably not sit comfortably even on the sound side. The patient will also suffer pain when standing upright. When we recall the relations of nerves to the joint, we can say that sacro-iliac disease might reveal itself by pain passing along the distribution of the obturator nerve which passes over the front of the joint. The great sciatic nerve sends nerves to the sacro-iliac joint, and may give remote symptoms of pain in the hip-joint or knee-joint, or back part of the calf of the leg. The superior gluteal nerve lies close to the sacro-iliac joint, and supplies the deep glutei muscles and tensor vaginae femoris, and might produce spasm or wasting of these muscles. Due to irritation, the psoas magnus would be contracted, and would produce a flexed condition of the thigh. We might, therefore, have a flexed condition of the hip-joint or thigh, inability to stand on the leg, or to sit comfortably, pain in the knee, pain in the leg, wasting of the gluteal region, or flattening of the muscles, and all these symptoms depending upon disease of the sacro-iliac joint.

One can see that it is practically impossible to diagnose accurately in the living subject between disease in the lower lumbar vertebrae and the sacrum, and between the sacrum and the upper part of the os innominatum, without a complete and thorough x-ray examination of spine, pelvis and hips. This will often show the exact site of disease. Most of our success in treatment depends upon accurate diagnosis. There is no question that disease between the sacrum and ilium is serious, and many physicians think it to be almost incurable, if of a serious nature. This, however, is not so, particularly if the disease be detected early.

In disease in the neighborhood of the sacro-iliac joint or hip-joint we should never forget to make a rectal examination, so as to find the presence or absence of abscess or tenderness.

Abscesses will frequently be completely absorbed, and will disappear, when the diseased areas are put at complete mechanical rest. The treatment of these conditions is simple, but must be continued sometimes for many months or even years.

The "Knee Flexion Test for Pathology in the Lumbosacral and Sacro-iliac Joints," as described by I. William Nachlas, is of distinct help in diagnosing conditions about these areas. We know that low back pain may be due to involvement of the gynecological, genito-urinary, gastro-intestinal and retroperitoneal tracts, as well as involvement of the skeletal tract. The simple test which the author offers, by virtue of its remote control, avoids the confusion that attends direct manipulations of structures near the affected area.

In this test the patient lies relaxed in a prone position on a rigid examining table, and the examiner flexes the knee by raising the foot up from the table. If the test be positive, the patient feels pain in the sacro-iliac or lumbosacral region, and at times along the nerves that run in front of these joints. The radiating pain follows the course either of the sciatic nerve, along the back of the leg to the calf, or the external cutaneous nerve of the thigh, on the outer side of the thigh to the knee. If the patient has no skeletal pathology in the lower part of the back, he will feel no pain, or only a feeling of tension in the front of the thigh.

The possibilities for error in technique are few. The necessary precautions are: (1) the table should be flat and non-yielding; (2) the patient should lie prone and relaxed, with the legs side by side; and (3) the leg should be passively raised directly up to flex the knee. It is advisable to place a hand lightly on the back to keep the patient from buckling at the hips.

Dr. Nachlas has used this test on hundreds of cases and has concluded that a positive test indicates disease in the lower part of the back, and when a specific joint responds, that is the affected one.

SUGGESTIONS ON TREATMENT

Immobilization and general constitutional care are two very important measures in the treatment of tuberculosis of the spine. Improvement of the disease in the spine usually follows improvement in the general condition of the patient. Poor health affects the patient adversely. Rest treatment is the most important single measure to bring about arrest of tuberculosis. Local repair is brought about by immobilization of the spine either by external or internal procedures. Internal fixation is employed with patients not suffering from active pulmonary tuberculosis. Active far advanced pulmonary tuberculosis was present in all my cases except one, and external immobilization had to be used. When greater stability of the spine is necessary internal fixation is employed. It is produced by the formation of a bridge of bone along the spinous processes about the area of disease. The two outstanding methods of internal surgical procedures are Albee's bone grafting and Hibbs' spinal fusion, and the various modifications of these operations.

The mobility of the spine is important both in diagnosis and treatment. It depends chiefly on two factors: the small intervertebral joints and the intervertebral discs. It is also affected by the spinous processes and the ligaments which bind them together, as well as the ligamenta flava which intervene between adjoining laminae. Any interference in the free movement of these structures leads to a disturbance in the mobility of the spine. Any hindrance causes limitation, partial or complete, of the movements of the area

of involvement of the spine. The movements of the entire spine may be only slightly disturbed in spite of stiffening of the affected segment, because the movements of the spinal column as a whole depend upon the sum total of the mobility of the various segments of the spine.

When new bone formation has begun on or about the diseased vertebral bodies and the small intervertebral joints and ligamenta flava, internal fixation either by means of the Albee bone grafting or Hibbs' spinal fusion so as to stabilize the diseased area of the spine may seem superfluous. The indication still further loses its value when the bone lippings have reached such size and form as to cause a more or less complete natural immobilization of the diseased area of the spine. This natural attempt at stabilization through ankylosis of the joints involved between the bodies of the vertebrae and the small articulations of the articular processes of the spine, as well as ossification of the ligaments included in the area involved, is a common manifestation of the processes of repair of the disease of the vertebra.

Bony union between two or more adjacent vertebral bodies with new bone formation about the posterior arch does not always signify complete healing in the vertebral lesion. Sometimes when the bony fusion seems by x-ray to be complete, active tuberculous foci which are not seen by x-ray, are found in the area on pathological examination. This fact is important not only in the treatment, but also in prognosis of cases of tuberculosis of the spine.

Many cases of tuberculosis of the spine are seen which are allowed part activity even in the early stages, and with support that is inadequate. Many cases also of early active pulmonary tuberculosis are allowed part activity. We believe that both these two conditions, occurring either in the spine or in the lungs should always be treated seriously, not only to arrest and cure the disease, but to prevent extension and destruction of tissue. When, therefore, we are dealing with active tuberculosis of the spine we always advise complete bed rest treatment. Even bathroom privileges are almost always denied. These patients receive nursing care. They are placed on a board mattress or a Bradford frame, and when necessary a plaster of Paris cast is applied. This complete bed rest is continued until we are reasonably sure that the spine is again strong. The cast is then changed to a brace, and the brace is worn indefinitely. Sometimes the patient is kept in bed in the cast for a period of five years.

With regard to fusion for those patients who are not suffering from pulmonary tuberculosis, there are what might be called two schools of thought, the so-called radical, and the conservative. It would seem that surgery is indicated when the lesion is well on the road to recovery or completely healed, and the reformed bone will

not be sufficient to give adequate support to prevent deformity, or would tend to allow a recurrence. Some orthopoedists advise surgery in about every case, but this advice is not universally accepted, even if there be only a lesion in the bone.

When we have placed our patient with tuberculosis of the spine under the most favorable conditions to produce rest to the spine we find that pain and muscle spasm are lessened. When the bone is actually in the stage of necrosis or destruction, or even reformation, it is soft and will be crushed at least partly by weight bearing.

In addition to the pain in the spine we usually detect some degree of muscle spasm and restricted motion; and dependent on the involvement the knee jerks will show an increase while the disease is active.

In the hip it is usually possible to elicit slight degrees of spasm even in the early stages.

CASE REPORTS

Miss J. C., age 30, white, consulted me May 9, 1926, for bilateral pulmonary tuberculosis and a pleurisy with effusion in the left chest. She had no cough and no expectoration.

She was seen in March, 1921, by her orthopoedist, and was then suffering from soreness in the region of the right great trochanter. The examination showed a tender spot in this region, and the x-ray showed a small area of inflammatory bone on the outer side of the great trochanter. In April, 1921, the surgeon cut down on the great trochanter and split the tendon of the gluteus medius. He at once dropped into a large sub-gluteal bursa which contained a lot of curious fluid and a great deal of granulation tissue which looked tuberculous. He dissected out the whole bursa, and the pathologist reported: "Large areas of caseation surrounded by a zone of endothelial cells with giant cells. Diagnosis—tuberculous bursitis."

She was again seen by the surgeon in June, 1925, when she complained that for the past six months she had been having some low backache and aching down the outer side of the thigh to the knee. She limped slightly like a patient suffering from sacro-iliac disease. She had no tenderness anywhere, and the region of the great trochanter seemed to be quiet. X-ray examination showed nothing abnormal. At this time the surgeon was anxious about what these symptoms might mean, and was suspicious of commencing tuberculosis of the spine.

In May of 1926, when she first consulted me, there was slight tenderness of the right sacro-iliac joint; movements of the legs and spine were normal. She stated that since her operation in April, 1921, she had felt slight pain down the front of the right leg to the knee. This was not severe; also a drawing pain in the right hip. She also felt pain in the right sacro-iliac joint when lying on her back. This pain was not felt when she stood.

X-ray examination as of July 4, 1926, showed definite erosion of the right trochanter; also a little more irregularity in the right sacro-iliac joint as compared with the left. The change in the sacro-iliac joint, however, was not characteristic enough to warrant any definite statement.

At this time I was therefore dealing with a patient who had bilateral pulmonary tuberculosis, with pleurisy with effusion in the left chest, with erosion of the right great trochanter, with localized distinct tenderness

over the right sacro-iliac joint, with pain radiating from the area of the right hip down the front of the leg to the knee, and also pain in the right sacro-iliac joint. X-ray examination of the right sacro-iliac joint was not conclusive, but I felt reasonably sure that disease also existed there. We therefore decided to immediately put her under treatment not only for her lung, but for her hip and sacro-iliac disease, and she was put on a Bradford frame and not allowed to leave her bed. No plaster of Paris was used, but she was kept constantly flat on her back. She remained on the Bradford frame, and was not allowed bathroom privileges for one and a half years, when she was allowed to move around a little, and was given a corset. On July 23, 1928, she showed the following condition: Both lungs showed a well arrested tuberculosis; the spine and hip symptoms seemed well, and there were no subjective symptoms. She walked naturally and there was no pain. There was still slight tenderness over the crest of the right ilium and at right sacro-iliac joint, and over the right great trochanter; also slight tenderness over the lower lumbar region. There was also slight flattening of the right buttock. She wore a brace.

In June of 1930 there was no tenderness or soreness over the right hip; movements were all normal; there was slight flattening of the right buttock. She felt well and walked naturally, and the lung condition was well arrested.

Case 2—I. C. C., male, white, age 27, mining engineer. In June, 1918, active tuberculosis in apex of right lung was diagnosed. He consulted me July 12, 1920, and the following condition was found: Right lung—tuberculous infiltration throughout with beginning cavity formation. Left lung—tuberculous infiltration throughout. X-ray examination of intestines showed ulceration of the caecum. Tubercle bacilli were in his sputum.

In November, 1920, he complained of pain in region of right hip, which radiated down to the knee and to the foot. This was of the nature of a sciatic pain, and was intermittent. At times there was a throbbing pain in the region of the right hip. In May, 1921, he had lumbago. In June, 1921, there was pain in the right hip, right groin and right lower back. The pain was increased with coughing and on standing after sitting for some time. There was stiffness in the back in the morning. Examination showed: Forward bend limited, as also left lateral; posture symmetrical except he prefers to bear weight on left leg; no spinal deformity; no tender spines; moderate tenderness over upper part of right sacro-iliac joint and over gluteal part of sciatic nerve; hip-joints free; no pain on concussion; x-ray negative. At this time, June, 1921, a diagnosis of right sacro-iliac strain was made. Only front to back films of the spine were made, as lateral views had not yet been employed.

While we were not satisfied with the negative examination, we gave him treatment for a possible sciatica. No result occurred.

In November, 1921, his symptoms still persisted, and further x-rays were made. This time lateral views showed a well defined area of tuberculosis on the anterior portion of the fourth lumbar vertebra. There was also an abscess in the right groin. He was put on a Bradford frame and in plaster of Paris. The abscess was tapped on several occasions and finally disappeared. He remained in the plaster and on the frame for five years. At the end of this time he had no cough and no expectoration, the cavities had disappeared from the lungs, and the lungs were in a well arrested condition; his intestinal tuberculosis was healed; the spine showed an

area of disease on the anterior portion of the fourth lumbar vertebra, and a bridge of bone united the third and fourth lumbar vertebrae. He was given a brace for the spine and has remained well since.

In this case the clinical symptoms pointed to tuberculous disease in the lower spine, and we made a mistake in not instituting treatment at an earlier date, due to the fact that we relied on the x-ray findings and not on the clinical symptoms. Lateral views of the spine made the diagnosis.

Case 3—Mrs. A. S. P. first consulted me in May, 1928, and then had tuberculosis in the upper third of both lungs with a small cavity in the right lung. She also had tuberculous ulceration involving the ileo-caecal region and probably terminal ileum. Tubercle bacilli were in her sputum.

In February, 1927, she first felt pain in the left hip. This pain lasted off and on until July, and bothered her a great deal to sit or lie on the left side. In November, 1928, there was a hollow condition back of the right great trochanter; movements of the spine were limited and there was fear to move. There was no tenderness anywhere. She was unable to lie on the left side. At night there was pain from the left hip down to the inside of the knee, and to the outside of the heel. March 3, 1930, showed the left buttock fuller than the right, and there was definite tenderness over the left sacro-iliac joint. X-ray examination as of June, 1928, showed a slight rather indefinite irregularity on the left sacro-iliac articulation. The change, however, was not characteristic enough to warrant a statement that a definte pathological condition existed there.

On March 11, 1930, further x-rays of spine and pelvis were made, and these films revealed slight irregularity of the left sacro-iliac articulation, with rarefaction in this region. This rarefaction appeared to be more pronounced than in the films of June, 1928, and was sufficient to warrant the statement that a definite change existed in this region.

In view of the fact that this patient had bilateral lung tuberculosis and intestinal ulceration, a diagnosis of left sacro-iliac disease was made, and she was given a steel brace and put to bed on a board mattress. She had complete bed rest treatment for four years. In 1933 she seemed to be well. There was no tenderness over the spine or pelvis, or anywhere else, movements of the spine were all normal; there was no pain and no tenderness anywhere. She has remained well. The intestinal ulceration became cured, the disease in the lungs became arrested, and she became permanently free of cough and expectoration.

Case 4—C. McK., female, typist, age 28. First consulted me in August, 1930. At this time she had advanced tuberculosis of the right lung, with cavity in the lower third, also advanced tuberculosis in the left lung with multilocular cavity formation in the upper third. There was also tuberculous ulceration of the ileo-caecal region and terminal loops of the ileum. There was also narrowing of the intervertebral disc between the third and fourth lumbar vertebrae, with a definite area of disease involving the body of the third lumbar vertebra. Tubercle bacilli were present in the sputum.

Pain in the back began in August, 1929. It was worse at night, and radiated from lumbar region to hips and down the legs. The muscles showed no rigidity; the contour of the spine was normal and there was no deformity. There was moderate tenderness of thro and thro type at sixth dorsal vertebra, and marked thro and thro tenderness at level of first and second lumbar vertebrae; no root tenderness; no bulging of soft tissues. The abdomen was clear, though there was a little resistance on the left

side, but no abscess was palpable. Knee jerks were normal. No Babinski; no clonus; no sensation of numbness in toes.

X-ray examination of the entire spine, pelvis and hips—front and lateral views and in stereo where possible, showed: a definite area of disease involving the body of the third lumbar vertebra. There is also a narrowing of the intervertebral disc between the third and fourth lumbar vertebra with a roughening of the articular surface. There is a questionable narrowing of the intervertebral disc between the fourth and fifth dorsal vertebrae.

At this time there was tenderness from the fifth to eighth dorsal vertebrae, and from the twelfth to the third lumbar vertebrae—marked over lumbar; movements were limited and stiff; the right leg flexed with pain radiating to hip and back; the left leg flexed with slight pain.

She was put on a board matress and in a plaster of Paris jacket from neck to pelvis, August, 1930. In August, 1940, the x-ray examination showed evidence of fusion of the third and fourth lumbar vertebrae. It also showed evidence of organization or possible calcification of what might previously have been abscess formation in the psoas muscle region.

At this time, 1940, both lungs showed a well arrested condition with absence of cavitation. No tubercle bacilli were in sputum. Cough and sputum were slight and often absent. She weighed 165 pounds, and felt and looked to be in good health. On May 29, 1942, the spine showed no deformity. There was slight tenderness on deep pressure at third or fourth dorsal vertebrae, and at third or fourth lumbar vertebrae; forward bend is limited, also lateral bends, especially to the left; knee reflexes exaggerated.

Case 5—Mrs. F. B. J., age 28, housewife, consulted me in April, 1930. She had tuberculosis throughout the right lung, with cavity in the upper third, also tuberculosis throughout the upper two thirds of left lung. She also had tuberculous ulceration involving the ileo-caecal region and a portion of the ascending colon. There was also tuberculosis of the urinary tract with tubercle bacilli in the urine. There was also tuberculosis of the left sacro-iliac joint, left knee and left ankle. Tubercle bacilli were in her sputum. She was put into a plaster of Paris cast extending from well up above the hips down to the toe of the left foot. She remained in the cast for two and a half years, and also on a board mattress. At this time, two and a half years, she had no cough and no expectoration. Tuberculous ulceration in the intestines had healed. Tubercle bacilli had disappeared from the urine—by guinea pig inoculation. The cavity had disappeared from the lung, and both lungs showed a well arrested condition. The right buttock seemed to be a little fuller than the left. There was no pain or tenderness in the left sacro-iliac joint and the spine. Movements of the back and hips were normal. The left knee was a little fuller than the right and the contour was not so well marked, but there was no tenderness or stiffness. The left ankle was still a little thickened, but there was no pain or tenderness. She has remained well and without symptoms anywhere, and is now an office nurse in a doctor's office.

REFERENCES

1 Hilton, John: *Lectures on Rest and Pain*, G. Bell & Sons, Ltd., London, Eng., 1930, p. 91.
2 Alfer: "Die Haufigkeit der Knochen und Gelenktuberkulose im Beziehung auf Alter, Geschlechtusw." *Beitr. klin. Chir.*, 8: 277, 1892.
3 Vulpius: Cited by Konschegg, *Handbuch der speziellen pathol. Anatomie und Histologie*, Lubarsch und Henke, J. Springer, 1934.

4 Nathanson, L., and Cohen, W.: "A Statistical and Roentgenological Analysis of Two Hundred Cases of Bone and Joint Tuberculosis," *Quart. Bull., Sea View Hosp.*, 6: 148, (Jan.) 1941.
5 Auerbach, O.: "Tuberculosis of the Skeletal System," *Quart. Bull., Sea View Hosp.*, 6: 117, (Jan.) 1941.
6 Harris, R. I., and Coulthard, H. S.: "End Results of Treatment of Potts' Disease," *Jour. Bone and Joint Surg.*, 22: 862, (July) 1940.
7 Sweet, P.; Bennett, G., and Sweet, D.: "Potts' Disease; The Initial Lesion. The Relative Infrequency of Extension by Contiguity. The Nature and Type of Healing. The Role of the Abscess, and the Merits of Operative and Non-Operative Treatment," *Bone and Joint Surgery*, 22: 878, (July) 1940.
8 Krause, F.: "Die Tuberkulose der Knochen und Gelenke," *Dtsch. Chir.*, 1899.
9 Konig, F.: *Die Spezielle Tuberkulose der Knochen und Gelenke*, Berlin, Vol. I, 1896, Vol. II, 1902.
10 Lexer, E.: *Weitere Untersuchungen der Knochenarterien und ihre Bedeutung fur Krankhafte Vorgange*, 1904.
11 Schmidt, M. B.: *Erkrankungen der Bewegungsapparats. Aschoff's Lehrbuch der pathologischen Anatomie*, Jena, 1928.
12 Boyd, W.: *A Textbook of Pathology*, Lea & Febiger, Philadelphia, 1930.
13 MacCallum, W. G.: *A Textbook of Pathology*, W. B. Saunders Co., Philadelphia, 1930.
14 Santos, J. V. de los: "Tuberculosis of the Spine," *The Bulletin of The Quezon Institute*, Vol. 1, No. 4, (Jan.) 1941.
15 Doub, H. P., and Badgley, C. E.: "The Roentgen Signs of Tuberculosis of the Vertebral Body," *Amer. Jour. Roent. & Rad. Ther.*, 27: 827-834, 1932.
16 Nachlas, I. William: "The Knee Flexion Test for Pathology in the Lumbo-Sacral and Sacro-iliac Joints," *The Jour. of Bone and Joint Surg.*, 18: 724, 1930.
17 *Gray's Anatomy.*
18 Morris: *Human Anatomy.*

Tuberculous Tracheobronchitis*

DAVID McCULLOUGH, M.D., F.C.C.P.**
Sanatorium, Texas

Only within the past ten years or so has the attention of physicians treating tuberculosis been drawn to the problem of tuberculous tracheobronchitis, despite the fact that the pathological findings were clearly described many years before. Since the early publications of Eloesser[1] and Coryllos[2] a steadily increasing number of papers dealing with this subject have appeared. No attempt will be made to review this extensive literature. We propose to discuss briefly some of the salient aspects of the disease in the light of our own experience and that of others. It is to be regretted that little can be added which might make treatment more effective.

PATHOLOGY

Bronchial infection must have complicated the course of pulmonary tuberculosis in as many cases in the past, yet it is strange that to many of us the manifestations of this phase of the disease seem to have been encountered much more frequently in the last few years. Exact figures on incidence vary, though Alexander's[3] figure of eleven per cent seems to be representative. Our own figures are somewhat lower but bronchoscopy has been done in comparatively few cases and the diagnosis has been made largely on x-ray and clinical evidence. Whatever the incidence the complication occurs in a sufficiently large number to cause distinct worry to anyone treating tuberculosis.

The bronchoscopists and pathologists alike describe several forms of endobronchitis. The acute lesions may be ulcerative or hyperplastic, or a combination of the two. The fibrostenotic lesion represents the chronic end stage and stenosis may be slight or almost complete. It has long been noted that the smaller bronchi leading from pulmonary cavities are almost uniformly diseased, but the larger bronchi and trachea may be the site of disease varying from a slight area of mucosal redness to ulceration and marked fibrous stenosis. The lumen of the air passage may be narrowed by edema and exuberant granulation in the acute phase, by fibrous contraction in the chronic stage, and in still a third manner, according to Chamberlain and Gordon.[4] The latter state that the ulceration may

---

*Presented at the Annual Meeting, Texas, Chapter, American College of Chest Physicians, Houston, Texas, May, 1942.

**From the State Tuberculosis Sanatorium.

destroy part of the bronchial cartilage and that after healing occurs this portion of the bronchus may be relatively flaccid. During inspiration air will pass the area freely but during the phase of expiration a collapse of the bronchial wall may occur sufficient to impede the passage of air.

Thus the problem of tuberculous tracheobronchitis is largely one of mechanical obstruction and improper drainage of secretions distal to the diseased bronchus. Drainage is impaired not only by mechanical obstruction but by impairment of bronchial peristalsis and loss of ciliary action. Because of these changes patients who have this complication are apt to have obstructive emphysema or atelectasis and resulting bronchiectasis. The amount of pulmonary tissue involved will depend on the location of the endobronchial lesion. Somewhat comparable in its development to obstructive emphysema is the so-called "check-valve" cavity, frequently seen as a further complication. Most tuberculous involvement of the trachea and bronchi seems to follow cavity formation in the lung.

SYMPTOMS

Of the symptoms, severe, often paroxysmal, cough is one of the most common. The cough may be out of proportion to the amount of disease visible by x-ray. This is often associated with a marked difficulty in raising sputum. The amount of sputum may be scanty and mucoid for a time and then become abundant and purulent. The wide variation in the amount of sputum raised is quite suggestive. Persistent streaking is also rather commonly seen. A wheeze, audible to the patient, is very frequently noted. It is not uncommon to have patients tell us that for long periods of time they have been treated for asthma. Dyspnoea is not often severe but may be out of proportion to the amount of disease evident on examination. In only one of our cases has it been of serious proportions and in this case a rather marked stenosis of the trachea was present; the parenchymal lesion was limited to the upper third of the left lung. In those cases in which massive atelectasis appears dyspnoea may be notable, associated with temperature elevation and some pain or sense of contraction in the affected side of the chest. Febrile episodes, associated with retention of secretions, are often seen. Cyanosis may be present. In cases of long standing atelectasis followed by bronchiectasis signs of sepsis may be seen due to faulty drainage of secretions from the bronchiectatic sacs.

PHYSICAL FINDINGS

The physical signs are often absent, and if present will vary with the state of the lung distal to the obstruction. Rhonchi and wheezes are frequently heard, often without the stethoscope. Clicks can

sometimes be heard by listening with the ear placed just in front of the patient's open mouth. Areas of atelectasis, if sufficiently large, will usually produce characteristic bronchial breath sounds; in some cases no breath sounds will be heard. Over check-valve cavities it is often possible to hear a wheeze just at the end of both inspiration and expiration, though the characteristic cavity signs will be absent because of the poor bronchial communication. We believe that the signs and symptoms which have been mentioned are highly suggestive of endobronchitis, and that when suggestive x-ray findings are also present the diagnosis can be made with considerable accuracy. A positive diagnosis can be made only by bronchoscopy.

ROENTGENOLOGIC FINDINGS

A knowledge of the pathology will aid considerably in the interpretation of x-ray findings. Most of the roentgenologic findings are indicative of endobronchitis only indirectly, but they are nevertheless of great aid in the diagnosis. Among them are atelectasis, balloon or check-valve cavities, localized emphysema, sudden disappearance of a cavity and unexplained spread of disease.

The appearance on the film of atelectasis, either of a lobe or an entire lung, is usually quite typical. The characteristic ground glass opacity of the affected lobe or lung with homolateral displacement of the mediastinum, elevation of the diaphragm on the same side and narrowing of the interspaces make recognition easy. Its occurrence is always the result of bronchial occlusion and its development in a patient with pulmonary tuberculosis is strong evidence of a tuberculous bronchitis. Other factors may produce it, such as pressure from without the bronchus due to enlarged lymph nodes, the presence in the bronchial lumen of tenacious sputum, etc., but we feel that these are of minor importance in the etiology of atelectasis. Atelectasis of a lower lobe, especially on the left side, may be somewhat difficult to demonstrate but can usually be seen as a triangular area of density at the base, partly hidden by the heart shadow.

The presence of round or oval cavities, often with a fluid level, is highly suggestive of tuberculous bronchitis with obstruction of the draining bronchus. Cavities of this type often appear tense and distended; there is frequently very little evidence of infiltration in the surrounding lung field, and the cavity wall appears thin. If artificial pneumothorax is administered, the cavity may or may not close; very frequently these check-valve cavities fail to close regardless of the amount of collapse of the lung obtained. Indeed they quite often become distinctly larger as the lung is collapsed. We have seen more than one of them become so distended with air that rupture of the cavity wall occurred, followed by empyema. On the

other hand, a cavity of this type may remain open and constant in size, under pneumothorax, for weeks and then close suddenly. This can only be explained on the assumption that the bronchus has become completely occluded and the air within the cavity absorbed. Cavities of this type are often seen within an otherwise atelectatic lobe.

A bronchogenic spread of disease in a patient without demonstrable cavitation should make one suspicious of a bronchial infection and aspiration of purulent material from a tuberculous ulcer.

Localized emphysema is mentioned by some as a common occurrence when a check-valve mechanism exists in the bronchus. Such emphysema undoubtedly occurs; we have not been struck by its appearance in our films but quite possibly have overlooked it.

As direct roentgenologic evidence of tuberculous disease of the bronchus Oechsli[5] lists the following: encroachment on the lumen of a bronchus, found only in cases of gross involvement of the larger bronchi, sometimes demonstrable only after filling of the bronchus with an opaque medium; and thickening of the bronchial wall, occasionally visible when the bronchus is thickened but not completely occluded.

TREATMENT

Before any form of collapse therapy is undertaken in a patient whose signs or symptoms suggest a bronchial lesion bronchoscopy should be seriously considered. The treatment of pulmonary tuberculosis so complicated is frequently disappointing; the unfavorable influence of the bronchial lesions on the prognosis is well known. It is true that spontaneous healing of endobronchitis often occurs but it is also apparently true that healing may take place in one area at the same time that new lesions develop in another.

Local treatment through the bronchoscope is frequently very effective in giving relief from symptoms of obstruction. Edematous, hyperplastic or ulcero-hyperplastic areas may be shrunken by the application of cocaine and adrenalin; the reduction of the obstruction may be only temporary but may result in markedly increased drainage of secretions and relief from dyspnoea, cough and fever. The smooth fibrostenotic lesions can sometimes be dilated by careful bronchoscopy with the same results. It seems to be generally agreed that healing of the ulcerative form is accelerated by local application of 30 per cent silver nitrate.

The choice of treatment of parenchymal disease is often greatly influenced by the presence of bronchial disease, particularly bronchial stenosis. Our attempts to collapse check-valve cavities with pneumothorax have often failed and in a few cases have been disastrous. In some cases the cavity steadily increases in size despite

increasing collapse of the lung, and reexpansion of the lung has been done; in a few cases the cavity has reached giant proportions and ruptured before reexpansion could be accomplished and empyema has followed. In some cases, chiefly when the cavities were not large, closure of the cavity with atelectasis of the lobe have followed pneumothorax, often several weeks after collapse was begun. Most, though not all, of the blocked cavities which have closed with pneumothorax have been rather small and have been surrounded by considerable lung parenchyma. Our experience with large apical cavities, located near the periphery of the lung, has been almost uniformly disappointing.

Collapse by pneumothorax of a lung in which stenosis has produced massive atelectasis should probably not be done unless the symptoms resulting from displacement of the mediastinal structures are severe. Collapse will have little effect upon the already airless lung, but will restore the mediastinum to its normal position. It is doubtful that reexpansion of such a lung can be accomplished and permanent collapse will probably be necessary.

We believe that the presence of endobronchial lesions which do not produce stenosis should not deter us from the use of pneumothorax when the parenchymal lesion indicates it.

By and large, we can fully agree with others that pneumothorax in the treatment of tuberculosis complicated by stenotic bronchial lesions is very unsatisfactory.

In many cases of large blocked cavity cavernostomy by the Monaldi method of drainage may give marked relief from symptoms. Cough will frequently all but disappear, the temperature will fall and the general condition of the patient may greatly improve. It seems, however, that the end results of Monaldi drainage are disappointing in the great majority of cases; few permanent cavity closures are reported.

Lobectomy and even pneumonectomy have been advocated in cases of stenotic endobronchitis. Rarely are suitable cases for pneumonectomy found; any disease in the contralateral lung, no matter how slight, is a definite contraindication, and the mortality is high. Lobectomy may prove to be feasible when the disease is largely limited to one lobe. If the bronchus can be divided proximal to the area of endobronchitis, it seems that favorable results should be expected. Few such cases, followed for a sufficient time, have been reported, and the results are not encouraging.

Although it does not seem logical to think that thoracoplasty would be apt to close a blocked cavity, there are many reports of the successful use of this procedure in such cases. The consensus of surgeons seems to be that thoracoplasty is the treatment of choice of both blocked cavities and atelectasis resulting from stenotic en-

dobronchitis. In some cases cavity drainage, either before, during or after operation, may be necessary to obtain cavity closure. Bronchoscopic drainage in the immediate post-operative period is frequently indicated when cough is not sufficient to result in adequate drainage.

SUMMARY AND CONCLUSIONS

1) About eleven per cent of cases of tuberculosis of the lung parenchyma are complicated by tuberculous tracheobronchitis.

2) Acute endobronchitis may be of the ulcerative type, the hyperplastic type or a mixture of both. The smooth, fibrostenotic lesion represents the chronic stage.

3) The problem of tuberculous tracheobronchitis is largely one of mechanical obstruction and improper drainage of secretions.

4) The chief symptoms are cough, often severe and paroxysmal, wheezing, asthma-like attacks, fever of an intermittent type and dyspnoea.

5) The significant physical signs are rhonchi and wheezing, plus signs of atelectasis in cases with this complication.

6) The most common x-ray findings are those of atelectasis, and check-valve or balloon cavity with a fluid level.

7) The diagnosis can be made with certainty only by bronchoscopy. A negative bronchoscopic examination does not eliminate the possibility of disease of the smaller bronchi.

8) As a general rule the treatment of pulmonary tuberculosis complicated by endobronchitis without stenosis should be that of a similar lesion not so complicated; if ulceration is present, local treatment with 30 per cent silver nitrate is of additional benefit. For cases complicated by stenotic bronchial lesions thoracoplasty is the procedure of choice; local treatment through the bronchoscope of the stenotic lesion itself is often necessary as a supplementary or complementary procedure. Aspiration and drainage of blocked cavities may be indicated before, during or after thoracoplasty. Pneumothorax in the treatment of tuberculosis complicated by stenotic bronchial lesions gives disappointing results in the great majority of cases and its use is not without danger.

RESUMEN Y CONCLUSIONES

1) Un once por ciento de los casos de tuberculosis del parénquima pulmonar sufren de la complicación de traqueobronquitis tuberculosa.

2) La endobronquitis aguda puede ser del tipo ulcerativo, del hiperplástico o de una mezcla de ambos. La lesión fibroestenótica lisa representa el período crónico.

3) El problema que presenta la traqueobronquitis tuberculosa

consiste principalmente en que causa obstrucción mecánica y drenaje inadecuado de las secreciones.

4) Los síntomas principales son: tos, que es a menudo severa y paroxismal, respiración ruda, ataques semejantes al asma, fiebre de tipo intermitente y disnea.

5) Los signos físicos significativos son: estertores roncantes y sibilantes, más los signos de la atelectasia en casos que presentan esta complicación.

6) Los hallazgos radiográficos más frecuentes son los de la atelectasia y cavernas de tipo de válvula de retención con un nivel de líquido retenido.

7) Sólo puede hacerse con certeza el diagnóstico mediante la broncoscopia; pero un examen broncoscópico negativo no elimina la posibilidad de la existencia de la enfermedad en los bronquios menores.

8) Por regla general, el tratamiento de la tuberculosis pulmonar con la complicación de la endobronquitis sin estenosis, debe ser lo mismo que en el caso de una lesión similar sin tal complicación; si existe ulceración el tratamiento local con una solución de 30 por ciento de nitrato de plata es también beneficioso. En casos con lesiones bronquiales estenóticas la toracoplastia es el procedimiento de elección; el tratamiento local de la lesión estenótica misma mediante el broncoscopio es a menudo un procedimiento suplementario o complementario necesario. La indicación para la aspiración y drenaje de las cavernas bloqueadas puede ocurrir antes, durante o después de la toracoplastia. En la gran mayoría de los casos el neumotórax no da buenos resultados en el tratamiento de la tuberculosis complicada por lesiones bronquiales estenóticas y su empleo puede ser perjudicial.

REFERENCES

1 Eloesser, L.: "Bronchial Stenosis in Pulmonary Tuberculosis," *Am. Rev. Tuberc.*, 30: 123, (Aug.) 1934.

2 Coryllos, P. N.: "The Importance of Atelectasis in Pulmonary Tuberculosis," *Am. Rev. Tuberc.*, 28: 1, (July) 1933.

3 Alexander, J.; Sommer, G. N. J., and Ehler, A. A.: "Effect of Thoracoplasty Upon Pulmonary Tuberculosis Complicated by Stenotic Tuberculous Bronchitis," *Jour. Thoracic Surg.*, 11: 308, (Feb.) 1942.

4 Chamberlain, J. M., and Gordon, J.: "Treatment of Endobronchial Tuberculosis," *Jour. Thoracic Surg.*, 11: 292, (Feb.) 1942.

5 Oechsli, W. R.: "Tuberculous Tracheobronchitis," *Am. Jour. Roentgen.*, 46: 312, (Sept.) 1941.

Traumatic Pneumothorax Without Gross Injury To the Chest Wall (Case Report)

FRANK H. WASHBURN, M.D., F.A.C.S., F.C.C.P.
Holden, Massachusetts

Aside from the voluminous literature upon pneumothorax, spontaneous or otherwise, in its relation to pulmonary tuberculosis, much has been written regarding spontaneous pneumothorax in which no pulmonary disease could be determined.

Traumatic pneumothorax accompanying gross injuries to the chest wall, including gunshot and stab wounds, depressed fractures of the bony thoracic cage, etc., has claimed considerable space in the medical press.

A less heralded but equally interesting group includes traumatic pneumothorax where preexisting lung and pleural lesions are not present and where also no appreciable injury to the thoracic wall occurs. Butler,[1] speaking of pneumothorax, states "The most dangerous type is that resulting from rupture of the lung without any demonstrable injury of the rib cage."

C. F. Hegner[2] states "Blunt traumatizing compressing force, especially on elastic chests of the young, can and often does cause extensive or fatal internal injuries without the slightest evidence of trauma to the superficial structures. The visible trauma to the thoracic wall is no index to the character or degree of intra-thoracic damage."

Harrington[3] of the Mayo Clinic said in discussing the subject "—serious injury and rupture of the underlying viscera can occur even though there is not an open wound."

Elkin and Harris[4] write "Forced expiration with the glottis closed raises the intra-pulmonic pressure. A crushing pressure applied to the chest as may happen in accidents, can raise it to such an extent that one or both lungs may be ruptured, even though the thorax is not penetrated."

It is accepted as fact that injury to the lung involving pneumothorax may occur by contre-coup whereby the lung contralateral to the side upon which the force was applied may receive apparently all or the grosser injury.

The following case is an illustration of lung rupture without gross chest wall injury and is interesting in that the pneumothorax was entirely missed for nearly three years.

Received for publication October 2, 1942.

Case—H. F. S., male, 45, married, automobile salesman, admitted to Holden District Hospital, Sept. 8, 1941, having been referred by Dr. Herman D. Stevens. His chief complaints were pain in chest, dyspnoea, and cough. He also mentioned pain in the lower right abdominal area and back. He gave the following history: On March 18, 1938, having delivered an automobile to a customer, he attempted to assist in pushing the car off an icy surface. In the process, he was spun around and fell to the ground. He tried to get to the garage nearby, but succeeded in only getting seated upon the running board of the automobile where he was obliged to remain an hour because of the immediate disability. He had extreme dyspnoea and felt as though he would "never get his breath." He was nauseated, had a stabbing pain in the right hemithorax and became drenched with perspiration. After an hour, he "gained his legs" and got into his car experiencing pain in the whole right side, especially the abdomen. Later the pain spread over the chest. He had no cough at this time but developed one later. He was driven to his place of employment and then taken to a surgeon employed by the liability company by whom his employer was insured. Thence he was removed to his home where he spent the next two months partly in bed, but not continuously so. About two weeks after the injury, he was taken to a urologist who cystoscoped him and reported negative findings. In May, 1938, he was advised to go to a hospital for application of a plaster cast. He had had "leg buckling" and at times pain in the right testicle. Instead of going to the hospital as advised, he consulted an osteopath, Dr. S., who diagnosed fourth and fifth lumbar displacement and prescribed a belt. The lower spine was x-rayed but not the chest at this time. He called Dr. S. off and on up to January, 1941, having had a persistence of girdle-like pain in the lower chest, cough and dyspnoea. In January, 1941, Dr. S. fluoroscoped the thorax and noting cardiac displacement, referred him to Worcester County Sanatorium where a diagnosis of right pneumothorax was made. Some air was removed from the right pleural cavity by Dr. Herman Stevens and the patient was referred to Belmont Hospital, under whose province he came, and to which institution he was admitted the same day (January 12, 1941). He remained there until February 3, 1941, during which time many specimens of sputum were examined by direct smear and were consistently negative to tubercle bacilli. So, also was a twenty-four hour concentrate test, and cultures were likewise negative. X-ray examination corroborated the diagnosis of right pneumothorax. No air was removed while the patient was at Belmont Hospital. He left the hospital of his own initiative. He was again seen as an out-patient at Worcester County Sanatorium where air was removed at about fortnightly intervals. The procedure was done eleven times up to his admission to Holden District Hospital on September 8, 1941. On one occasion, oxygen lavage as practiced by Welkind and Herman[5,6] at Sea View Hospital was done.

The problem presented us on admission was that of a well-developed non-tuberculous male patient of forty-five, the subject of a right bronchopleural fistula and pneumothorax of traumatic origin of more than three years duration. Removal of air had been carried out a sufficient period to indicate that permanent relief could not be expected to result from that procedure alone. Bronchoscopy had been done on April 5, 1941, by Dr. Julius Tekelberg, who had introduced iodized oil and had had bronchograms made resulting in the information that no bronchial growth or other obstruction had been present. The late Dr. George Albee had made

a cardiological examination on June 11, 1941, taking electrocardiograms with negative organic pathological findings, though a left preponderance had been noted by him.

Among the various measures considered by us as offering a possibility of relief was the production of a plastic pleuritis with continued air aspiration, hoping thereby that the adhesions provoked would seal the pleural opening of the fistula and cause symphysis of the parietal and visceral pleural surfaces. The ever-present threat of pyogenic infection via the broncho-pleural fistula during induced irritative pleuritis looms in one's mind as something of a hazard even in the absence of broncho-pleural infection.

Numerous measures and the application of various substances have been attempted both experimentally in lower animals and therapeutically in humans for the production of pleural coalescence. Bethune[7] used successfully poudrage with iodized talc after experimentation with many substances. Singer et al,[8] Hanrahan, Adams and Klopstock[9] and others experimented with many substances and showed the comparative effectiveness of iodized talc over other tried substances. Their studies, however, bring out certain possible objections to the use of silicate. Mechanical irritation such as brushing the pleural surface with tape, gauze and cautery have been used with effect. Kupka and Wagner[10] used injections of the patient's own blood into the pleural cavity for the purpose of obliteration of the pleural space prior to surgical attack of lung abscess. Blood was first suggested by Lloveral of Barcelona for pleural obliteration. The foreign body reaction to blood in intrapleural hemorrhage, resulting in a variable amount of pleural symphysis has been a rather common observation. Suggestions for the cure of intractable pneumothorax have included lobectomy; however, one would hesitate to adopt this major procedure.

Of the conceivable and suggested methods, the intrapleural injection of the patient's own blood seemed to us to offer the least hazard and this operation was done September 9, 1941. Thoracoscopy was performed through the 5th interspace using the Unverecht instrument. All the parietal pleura and that over the middle and lower lobes appeared smooth and glistening. On the anterior aspect of the upper lobe surface was a raised circular disk of tissue of estimated diameter of 1.5 cm., yellowish in hue and having a central dimple. This was not probed, nor was any attempt made to irritate the surface mechanically. After thorough inspection, 60 cc. of the patient's own blood was withdrawn from the left median basilic vein and immediately injected through the cannula of the instrument into the pleural cavity. The patient appeared a little faint and phrenic interruption was deferred. The trochar wound was closed by means of a deep chromic suture.

On September 12, 1941, after removal of 400 cc. of air from the right pleural cavity, and removal of the thoracoscopy stitch, the phrenic nerve was approached above the right clavicle and interrupted by crushing, deeming that temporary diaphragmatic paralysis, only, was desirable.

Throughout his stay in the hospital, we attempted to keep the visceral pleura as near in contact with the mural surface as possible by frequent aspiration of air which was done by my associate, Dr. L. E. Mayo, using at first the Robinson Pneumothorax Apparatus, but later the Tomac Apparatus (gastro-evacuator) was found more suitable. For some period, we left a blunt needle in situ and frequent aspirations were made throughout the day and night. At first, a small amount of fluid appeared as shown by fluoroscopy and films, later increasing somewhat. Not until October 8 did we feel it best to do a second thoracoscopy when a few frail adhesions were noted anteriorly and posteriorly between the surface of the lower lobe and chest wall while the lateral surface appeared to be free.

On October 12, 1941, the withdrawal of air having brought the lung out to the parietal surface, except for small areas about the upper lobe, although only partially adherent, we felt that time should be given an opportunity to complete the closure. We, therefore, discharged the patient for continuance of air withdrawals by Dr. Stevens at the Sanatorium with the understanding that he was to return in a few weeks for further procedures if need be.

His whole course at Holden District Hospital was practically afebrile. No further withdrawals of air became necessary, and as shown by a skiagram taken at Worcester County Sanatorium, the air space became completely obliterated.

Roentgenographic examination, June 30, 1942, showed but a faint remaining trace of the original shadow, marking the scar of the pulmonary injury, and the action of the diaphragm was no longer paradoxical though the right leaf was still relatively high. The vital capacity had gradually increased, and was at 2500, and there was no dyspnoea. He was working ten hours daily in a firearms factory, seven days a week, and enjoying his new vocation.

CONCLUSION

The case illustrates the type of serious lung injury resulting from traumatism without the presence of external or thoracic cage wounds. It also illustrates how attention may be diverted from the lung by symptoms of other and less serious phases of traumatism. In this case the pneumothorax was overlooked for more than two years. The role of intra-pleural instillation of blood in producing a plastic pleuritis and ultimate cure is interesting.

CONCLUSIONES

Este caso ilustra el tipo de lesión grave del pulmón causada por traumatismo sin heridas concomitantes externas o de la caja torácica. También ilustra la manera como los síntomas resultantes de otros aspectos menos serios del traumatismo pueden distraer la atención del médico fuera del pulmón. En este caso no se notó el neumotórax por más de dos años. Es interesante notar el papel que desempeñó la instilación intrapleural de sangre en la producción de una pleuritis plástica y en conseguir al fin la curación.

Holden Clinic.

REFERENCES

1 Butler, E.: "Injuries of the Chest and Abdomen," *Surg., Gyn. & Obstet.*, 66: 448, (Feb. 15) 1938.

2 Hegner, C. F.: "Surgical Treatment of Injuries of the Chest," *Am. J. Surg.*, 47: 394, (Feb.) 1940.

3 Harrington, S. W.: "Wounds of the Chest and Abdomen," *Proc. Staff Meet., Mayo Clinic*, 15: 808, (Dec. 18) 1940.

4 Elkin, D. C., and Harris, M. H.: "Injuries to the Chest," *Ann. Surg.*, 103: 688, (May) 1941.

5 Welkind, A., and Herman, M.: "Rapid Expansion of the Lungs," *Quart. Bull., Sea View Hosp.*, Jan., 1939.

6 Ibid, Jan., 1941.

7 Bethune, Norman: "Pleural Poudrage," *J. Thor. Surg.*, 4: 251, (Feb.) 1935.

8 Singer, Jones, and Tragerman: "Aseptic Pleuritis Experimentally Produced," *J. Thor. Surg.*, 10: 251, (Feb.) 1941.

9 Hanrahan, Adams, and Klopstock: "The Role of Experimentally Produced Intrapleural Adhesions," etc., *J. Thor. Surg.*, 10: 284, (Feb.) 1941.

10 Kupka, Edw., and Wagner, Wm.: "Intentional Obliteration of Pleural Space by the Use of Blood," *Am. Rev. Tuberc.*, 43: 291, (Feb.) 1941.

COLLEGE NEWS

PROGRAM

COLLEGE SOUTHERN CHAPTER

Meeting Conjointly with the

SOUTHERN MEDICAL ASSOCIATION

The annual meeting of the Southern Medical Association will be held at Cincinnati, Ohio, November 16-18. The organizations that will meet conjointly with the Southern Medical Association, each of which will present a program, are: American Therapeutic Society; American Academy of Pediatrics, Region 2; American Society of Tropical Medicine; National Malaria Society; American Public Health Association, Southern Branch; and American College of Chest Physicians, Southern Chapter. Tuesday, November 16, will be known as Kentucky-Ohio Day, and the clinical sessions will be presented by physicians from the Campbell-Kenton County Medical Society of Kentucky and the Cincinnati Academy of Medicine. The Southern Medical Association has prepared an interesting program for Wednesday and Thursday, November 17 and 18.

College Program

Arrangements have been completed for the organization of the Southern Chapter of the American College of Chest Physicians and for the College participation in the annual meeting of the Southern Medical Association. The Hotel Gibson at Cincinnati has been assigned as College headquarters for the meeting by the officials of the Southern Medical Association. Members of the College who plan on attending the meeting are urged to make their reservations early.

Southern Chapter

The Southern Chapter of the College will be organized at a luncheon meeting to be held Thursday, November 18. In accordance with the resolution adopted at a meeting of College members at the time of the meeting of the Southern Medical Association, held in Richmond, Virginia, in 1942, a Governor's Committee has been appointed by the President of the College to make the necessary arrangements for the organization of the Southern Chapter.

Governor's Committee

Dr. Benjamin L. Brock, F.C.C.P., *Chairman*, Kentucky
Dr. L. O. Davenport, F.C.C.P., Alabama
Dr. J. D. Riley, F.C.C.P., Arkansas
Dr. M. J. Flipse, F.C.C.P., Florida
Dr. J. A. Redfearn, F.C.C.P., Georgia
Dr. Louis A. Monte, F.C.C.P., Louisiana
Dr. Victor F. Cullen, F.C.C.P., Maryland
Dr. John S. Harter, F.C.C.P., Mississippi
Dr. Herbert L. Mantz, F.C.C.P., Missouri
Dr. Louis Bonner, F.C.C.P., North Carolina
Dr. Robert Shepard, F.C.C.P., Oklahoma
Dr. Leo F. Hall, F.C.C.P., South Carolina
Dr. R. R. Crowe, F.C.C.P., Tennessee
Dr. C. J. Koerth, F.C.C.P., Texas

Dr. Dean B. Cole, F.C.C.P., Virginia
Dr. George R. Maxwell, F.C.C.P., West Virginia
Dr. Wm. D. Tewksbury, F.C.C.P., District of Columbia

A committee comprised of the Regents of the College in the Southern States has been appointed by the President to assist the Governors in the arrangements for the meeting.

Regent's Committee

Dr. Champ H. Holmes, F.C.C.P., *Chairman*, Georgia
Dr. Charles M. Hendricks, F.C.C.P., Texas
Dr. Karl Schaffle, F.C.C.P., North Carolina
Dr. H. I. Spector, F.C.C.P., Missouri
Dr. Paul A. Turner, F.C.C.P., Kentucky

A Nominating Committee, comprised of Dr. Charles M. Hendricks, F.C.C.P., Chairman, El Paso, Texas; Dr. Paul A. Turner, F.C.C.P., Louisville, Kentucky, and Dr. Erskine M. Chenault, F.C.C.P., Decatur, Alabama, has been appointed to bring in recommendations for officers of the Southern Chapter. Members in the southern states should send their recommendations for officers of the Southern Chapter to the committee at an early date.

The Presidents of each of the College Chapters have been invited by Dr. J. Winthrop Peabody, F.C.C.P., Washington, D. C., President of the College, to officiate in the organization of the Southern Chapter of the College.

A Constitution and By-Laws for the Southern Chapter will be adopted at this meeting, and all new members of the College in the southern states will be inducted into the Chapter.

Scientific Program

The following scientific program has been prepared by the Program Committee of the College and will be presented on Wednesday, November 17, and Thursday, November 18, at the meeting of the Southern Medical Association.

"Tuberculosis Among Children and Young Adults"
Dr. Chester A. Stewart, New Orleans, Louisiana

"Chest Diseases in the Aged"
Dr. Arnold S. Anderson, F.C.C.P., St. Petersburg, Florida

"Bronchoscopic Kodachrome Motion Pictures of Tracheal and Bronchial Tuberculosis"
Dr. Paul H. Holinger, F.C.C.P., and Dr. Ralph G. Rigby, Chicago, Illinois

"Lung Resection in Chronic Pulmonary Diseases"
Dr. Richard Davison, F.C.C.P., Chicago, Illinois

"The Occurrence of Pulmonary Tuberculosis in Supposedly Screened Selectees"
Col. Arden Freer, (MC), F.C.C.P., Washington, D. C.

"New Growths of the Chest"
Lt. Col. Carl Tempel, (MC), F.C.C.P., Washington, D. C.

"Tuberculosis as a Navy Problem"
Lt. Comdr. D. F. Smiley, (MC-V (S) USNR), Washington, D. C.

"A Study of Rejectees for Thoracic Abnormalities"
Dr. Wm. A. Hudson, F.C.C.P., and
Dr. David S. Brachman, F.C.C.P., Detroit, Michigan

"The Relative Importance of the Anatomic and Physiologic Concept in Tuberculosis"

Dr. J. D. Riley, F.C.C.P., State Sanatorium, Arkansas

"The Indications for Total Pneumonectomy"

Dr. Evarts A. Graham, F.A.C.S., St. Louis, Missouri

"The Tuberculosis Problem in Puerto Rico"

Dr. J. Rodriguez Pastor, F.C.C.P., San Juan, Puerto Rico

Scientific Program Committee

Dr. J. A. Myers, F.C.C.P., *Chairman*, Minneapolis, Minnesota
Dr. Wm. A. Hudson, F.C.C.P., Detroit, Michigan
Dr. C. Howard Marcy, F.C.C.P., Pittsburgh, Pennsylvania

Luncheon and Dinner Meeting

The Board of Regents and the Board of Governors of the College will have a dinner meeting at the Hotel Gibson, Wednesday night, November 17.

The officials of College Chapters will meet for luncheon on Thursday, November 18, at the time of the organization of the Southern Chapter.

The Ohio Chapter of the College will hold a business meeting and election of officers at a luncheon on November 17.

Local Arrangements Committee

Dr. John H. Skavlem, F.C.C.P., *Chairman*, Cincinnati, Ohio
Dr. Norbert S. Greene, F.C.C.P., Cleveland, Ohio
Dr. Paul M. Holmes, F.C.C.P., Toledo, Ohio
Dr. Elmer E. Kirkwood, F.C.C.P., Youngstown, Ohio
Dr. Louis Mark, F.C.C.P., Columbus, Ohio
Dr. Myron D. Miller, F.C.C.P., Columbus, Ohio
Dr. Joseph C. Placak, F.C.C.P., Cleveland, Ohio
Dr. Joseph B. Stocklen, F.C.C.P., East Cleveland, Ohio
Dr. Giles Wolverton, F.C.C.P., Dayton, Ohio

Local Reception Committee

Dr. D. W. Heusinkveld, F.C.C.P., *Chairman*, Cincinnati, Ohio
Dr. James N. Christiansen, F.C.C.P., Cincinnati, Ohio
Dr. Charles J. Farrell, F.C.C.P., Covington, Kentucky
Dr. Samuel Iglauer, Cincinnati, Ohio
Dr. Aaron J. Kanter, F.C.C.P., Cincinnati, Ohio
Dr. William H. Lippert, F.C.C.P., Cincinnati, Ohio
Dr. Louis B. Owens, F.C.C.P., Cincinnati, Ohio
Dr. Louis H. Senteff, F.C.C.P., Cincinnati, Ohio
Dr. Charles M. Siegel, F.C.C.P., Cincinnati, Ohio
Dr. Howard L. Stitt, Cincinnati, Ohio

CHAPTER MEETINGS

New York State Chapter

At the annual meeting of the New York State Chapter of the College held in conjunction with the meeting of the New York State Medical Society at Buffalo, New York, on May 6, 1943, the following resolution was introduced by Dr. Nelson W. Strohm, F.C.C.P., Buffalo, Governor of the College for New York State:

Be It Resolved: That the New York State Chapter of the College go on record as extending its congratulations to Dr. Ralph C. Matson, F.C.C.P., Editor of the College Journal, "Diseases of the Chest," for outstanding achievement in revising the College journal and advancing the standards of the material published in the journal which makes it one of the finest medical periodicals of our day.

This resolution was seconded by Dr. Arthur Q. Penta, F.C.C.P., Schenectady, and unanimously passed by the members of the Chapter present at the meeting.

It was agreed that the New York State Chapter of the College hold a scientific session in New York City during the month of October. Dr. James S. Edlin, F.C.C.P., President of the New York State Chapter, presided at the meeting and the following officers were elected for the ensuing year:

Dr. Arthur Q. Penta, F.C.C.P., Schenectady, President
Dr. James H. Donnelly, F.C.C.P., Buffalo, Vice-President
Dr. Lyman I. Thayer, F.C.C.P., Glens Falls, Secretary-Treasurer
(re-elected)

Fall Meeting, New York State Chapter

The Fall meeting of the New York State Chapter of the American College of Chest Physicians will be held at the Hotel Biltmore, New York City, on October 15, 1943. The following program has been arranged:

"A Simple Bronchopulmonary Nomenclature and Its Clinical Application"
Dr. Chevalier L. Jackson, F.C.C.P., Philadelphia, Pennsylvania
"Experiences with Pulmonary Resections in Pulmonary Tuberculosis"
Dr. Richard H. Overholt, F.C.C.P., Brookline, Massachusetts
"Rehabilitation of Military Personnel as Carried Out in the Army"
Brigadier General S. U. Marietta, F.C.C.P., Washington, D. C.
"Idiopathic Spontaneous Pneumothorax, Incidence and Pathogenesis"
Dr. George G. Ornstein, F.C.C.P., New York, New York

Dr. J. Winthrop Peabody, F.C.C.P., Washington, D. C., President of the College, will be the guest speaker at the luncheon meeting. Dr. James H. Donnelly, F.C.C.P., Buffalo, New York, Vice-President of the New York State Chapter, will preside at the morning session and Dr. Arthur Q. Penta, F.C.C.P., Schenectady, President of the New York State Chapter, will preside at the afternoon session.

The American Public Health Association will meet at New York City, October 12-14, 1943.

Indiana Chapter

The Indiana Chapter of the College will hold its annual meeting in connection with the annual meeting of the Indiana State Medical Association in Parlor B, Claypool Hotel, Indianapolis, Tuesday noon, September 28. Dr. Herman E. Hilleboe, F.C.C.P., Senior Surgeon of the U. S. Public Health Service and Coast Guard, will be the guest speaker. His subject will be "Tuberculosis Control in Industry." He will also show some 35 mm. x-ray films. Discussants will be Dr. Fred Wishard, Medical Director of Delco-Remy Corporation and Dr. Philip Becker, F.C.C.P., Superin-

tendent of the James Parramore Sanatorium, Crown Point, Indiana. Following the presentation of the paper, there will be an x-ray conference sponsored by the Indiana Chapter of the College.

The annual meeting of the Indiana State Medical Society this year will be a joint meeting with the Ninth Naval District and a number of the papers will be presented by medical officers from the Navy Department. For further particulars concerning the Indiana Chapter meeting, communicate with Dr. James H. Stygall, F.C.C.P., Regent of the College for the district, 707 Hume-Mansur Building, Indianapolis, Indiana.

Pennsylvania Chapter

The Pennsylvania Chapter of the College will hold its annual meeting in connection with the meeting of the Pennsylvania State Medical Society, at Philadelphia on Wednesday, October 6, 1943. In addition to the election of officers and other business to come before the Chapter, there will be an x-ray conference of interesting and unusual films. Members of the College are requested to bring to the meeting such films for exhibit and discussion. The meeting is to be held at the Bellevue-Stratford Hotel from 4:30 to 6:30 P. M. For further particulars concerning this meeting or the presentation of films, communicate with Dr. Edward Lebovitz, F.C.C.P., 412 Jenkins Building, Pittsburgh, Secretary-Treasurer of the Pennsylvania Chapter.

Michigan Chapter

The annual meeting of the Michigan Chapter of the College will be held at the Statler Hotel, Detroit, on September 23, 1943, in conjunction with the meeting of the Michigan State Medical Society. A scientific program has been prepared by the members of the College in Michigan and will be given in Parlor F of the Statler Hotel at 3:30 P. M. For further particulars concerning this meeting, communicate with Dr. Willard B. Howes, F.C.C.P., Detroit, Michigan, President of the Michigan Chapter.

Cuban Chapter

On June 22, 1943, the Cuban Chapter of the College held its regular monthly session at Havana, Cuba, and the following program was presented:

"Primary Tumor-like Cervical Lymph Node Tuberculosis in Children: Report on 9 Cases"

Dr. Teodosio Valledor, Havana

"Report of Clinical Cases of Bronchial Tuberculosis"

Drs. F. J. Menendez, F.C.C.P., and Ferrer Basnuevo, Havana

Drs. Alfredo Antonetti, F.C.C.P., Rufino Moreno, Ricardo Sanchez Acosta, and Antonio Navarrete, F.C.C.P., took part in the discussion.

Following the scientific program, Dr. R. Sanchez Acosta, Secretary-Treasurer of the Chapter, made the annual report on the activities of the Cuban Chapter for the year 1942-1943. Election of officers was then held and the following officers were elected for the year 1943-1944:

Dr. Teodosio Valledor Campos, Havana, President

Dr. O. Suarez de Bustamente, Havana, Vice-President

Dr. Francisco J. Menendez, F.C.C.P., Havana, Secretary-Treasurer

Puerto Rico Chapter

In a report prepared by the Puerto Rico Chapter of the College to the Hon. D. Chavez, President of the Congressional Committee Investigating Social Conditions in Puerto Rico, it was pointed out that tuberculosis was one of the leading causes of death in Puerto Rico and that it was responsible for nearly one-half of all of the deaths occurring in the island in persons between the ages of 15 and 34. It is reported that the death rate in Puerto Rico was 243 per 100,000 in 1941. This rate for 1941, extremely high as it is, represents the lowest mortality rate for tuberculosis in Puerto Rico in the last fifteen years. The average number of annual deaths from tuberculosis is 4700. The actual number of beds available, counting private institutions, is less than 1700. Plans are being made to obtain 3,000 additional beds in Puerto Rico for the care of the tuberculous. This movement has been endorsed by the Commissioner of Health and the Governor for the island.

The insular government is now spending $650,000 annually for the maintenance of 1500 beds now in use for pulmonary tuberculosis. It is estimated that the building and equipment of the additional beds would cost approximately $6,000,000 and that these beds could be maintained at an annual expense of $1,500,000.

An active campaign is being waged by the members of the Puerto Rico Chapter of the College to bring about this much needed improvement in the tuberculosis situation on the island and progress is being made.

The Fellows of the Cuban Chapter of the College have been elected Honorary Members of the Puerto Rico Chapter of the College as a token of recognition of the excellent work done by the members of the Cuban Chapter in fostering good fellowship among the chest specialists in all of the Americas. The Cuban Chapter was the first Chapter of the College organized outside of the United States.

Major Maurice J. Abrams (MC), F.C.C.P., Brewton, Alabama, was appointed the executive officer of a new general hospital in London, England. This hospital has been acquired by the United States Army from the British government under reverse lend-lease procedure. The hospital was redesigned by United States engineers and rebuilt by the British Ministry of Works. It is used mainly for the treatment of emergency cases and is open to members of the Army and Navy, merchant seamen, U. S. Embassy personnel, Wacs and American Red Cross workers. The hospital is equipped with the latest technical apparatus, mostly of American design. The general hospital equipment, however, is British. There is a dental clinic and x-ray department.

Dr. Raymond C. McKay, F.C.C.P., Cleveland, Ohio, has been promoted to associate clinical professor of medicine at the Western Reserve University School of Medicine, Cleveland, Ohio.

Among the guest speakers at the 102nd annual meeting of the Wisconsin State Medical Society, held at Milwaukee, Wisconsin, September 13-15, 1943, were Dr. Paul H. Holinger, F.C.C.P., Chicago, Illinois, subject: "The Infant Larynx," and Dr. Dean M. Lierle, Iowa City, Iowa, subject: "Acute Upper Respiratory Infections and Their Control."

Major Sidney E. Wolpaw, (MC), F.C.C.P., Cleveland, Ohio, addressed the Union County Medico-Dental Society, at Sturgis, Kentucky, on July 6, 1943. Major Wolpaw spoke on "The Diagnosis of Early Tuberculosis." He presented x-ray films to illustrate his subject.

Members of the Keokuk County Medical Society met in Sigourney, Iowa, Friday evening, August 20, at which time a scientific recording on "Chest Injuries" by Dr. Jerome R. Head, F.C.C.P., Chicago, Illinois, was presented to the Society.

Dr. Esther Rosencrantz, F.C.C.P., associate professor of medicine, University of California Medical School, San Francisco, California, was given a dinner at the Palace Hotel, San Francisco, on July 9, 1943, by her friends and associates to mark her retirement. She had been a member of the staff since 1913. Dr. Rosencrantz was also a lecturer in medical history and bibliography in the medical school. Dr. Rosencrantz is a collector of the works of Sir William Osler, whose pupil she was at the Johns Hopkins School of Medicine, Baltimore. Dr. Rosencrantz was presented at the dinner with six rare Osler items. She has given her entire collection to the University of California Medical School.

POSITIONS AVAILABLE

Resident physicians wanted for tuberculosis sanatorium in Colorado. For further particulars, address Box 106A, American College of Chest Physicians, 500 North Dearborn, Chicago 10, Illinois.

Resident physician wanted for tuberculosis hospital in New York. For further particulars, write Box 107A, American College of Chest Physicians, 500 North Dearborn, Chicago 10, Illinois.

Position available as chief resident of a tuberculosis sanatorium in California. Preferably under 36 years of age. The service includes diagnostic work, treatment and surgery for all chest diseases in the general hospital and large outpatient department. The hospital has 470 beds, 105 of which are for tuberculosis. Recent graduate preferred; experience in tuberculosis work desirable. Man with some physical handicap would be considered. Salary: $300 per month with maintenance. For further particulars, address Box 109A, American College of Chest Physicians, 500 North Dearborn, Chicago 10, Illinois.

Position available for an assistant physician in a 200 bed tuberculosis hospital situated in central Illinois. Experience in chest diseases helpful but not essential. Compensation commensurate with ability. For particulars write: Box 103A, American College of Chest Physicians, 500 North Deaborn, Chicago 10, Illinois.

Experienced chest specialists wanted for residency in Puerto Rico. For further particulars, address Box 111A, American College of Chest Physicians, 500 North Dearborn, Chicago 10, Illinois.

POSITIONS WANTED

Experienced physician in diseases of the chest would like to obtain a position as Medical Superintendent of a tuberculosis sanatorium. Administrative experience. Address: Box 203A, American College of Chest Physicians, 500 North Dearborn, Chicago 10, Illinois.

Physician with more than twenty years of experience in tuberculosis and diseases of the chest would like to obtain a position as Medical Director of a tuberculosis sanatorium. At present employed. Address: Box 204A, American College of Chest Physicians, 500 North Dearborn, Chicago 10, Illinois.